Journal
OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION

This issue is in two parts. This is Part I.

(Part 2—Official Business Proceedings—is mailed to AVMA members only)

Vol. 133 **December 1, 1958** **No. 11**

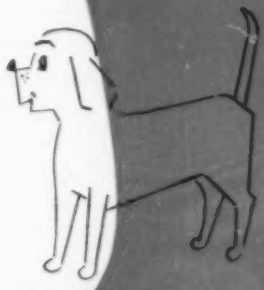
CSL

DIZAN*

dithiazanine iodide, CSL

Dizan Tablets offer the veterinarian a new, exclusive, broad coverage treatment for the common helminthic infestations of dogs and cats. Practitioners appreciate the safety and efficacy of Dizan, as it is gentle to animals, yet its removal of the worm burden approaches 100 per cent. Because Dizan is not a one-dose purger, there is an added advantage of not upsetting the schedule of pet or household. Dog breeders, too, will appreciate your mention of Dizan.

A 5 to 10-day treatment removes large roundworms, hookworms, and whipworms. Correct daily dosage is easily estimated — 10 mg. per pound of body weight given daily, preferably at or after feeding. Dizan is supplied in tablets of 50, 100, and 200 mg., packaged 100 tablets per bottle.

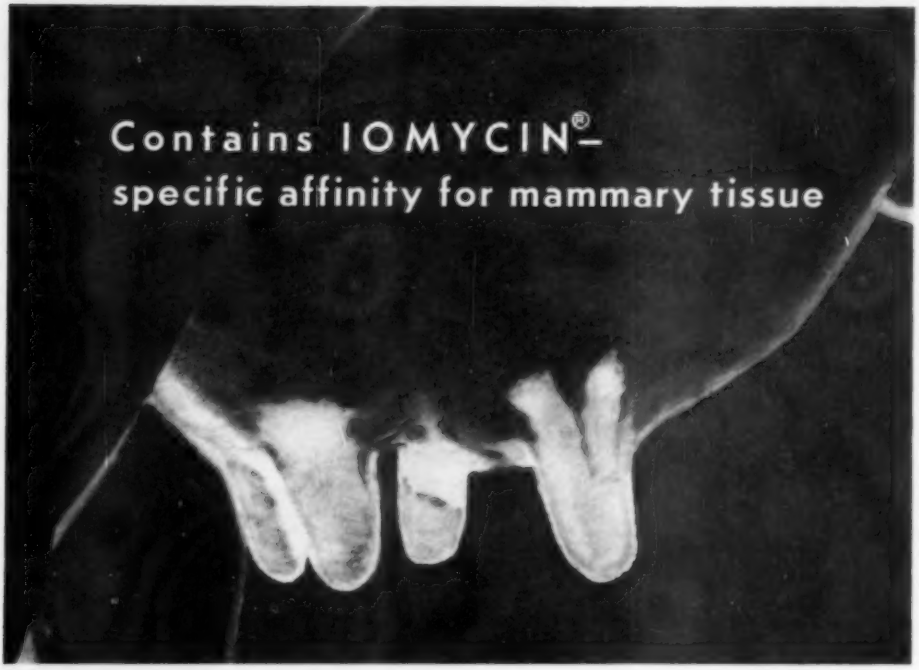


*T.M.

CSL

CORN STATES LABORATORIES, INC., 1124 HARNEY ST., OMAHA, NEBRASKA

QUADRAMYCIN



When mastitis calls for strong measures, dispense QUADRAMYCIN (4 antibiotics)

QUADRAMYCIN intramammary infusion contains **four** antibiotics . . . lomycin (DiethylaminoethylesterpenicillinGhydriodide with dihydrostreptomycin), polymixin B sulfate, and neomycin. This combination provides an exclusive professional formula for effective mastitis therapy.

QUADRAMYCIN features lomycin—the remarkable antibiotic that provides prolonged bactericidal activity . . . **plus** up to 5 times more udder tissue concentration than procaine penicillin G.

Display cartons of 12 syringes

Each 12 cc. QUADRAMYCIN syringe contains:
DiethylaminoethylesterpenicillinGhydriodide, 100,000 units
Dihydrostreptomycin (base as sulfate), 100 mg.,
Polymixin B sulfate, 50,000 units
Neomycin base (as sulfate), 100 mg. In special diffusible base.

Norden®
NORDEN LABORATORIES
Lincoln, Nebraska

Journal of the American Veterinary Medical Association

CONTENTS

Continued from Cover

SURGERY AND OBSTETRICS

CLINICAL DATA

NUTRITION

$15.00 per Annum Single Copies, $ .75 Prepaid in U.S.
Canada and Pan American Countries, $15.50; Foreign, $16.50
Published semimonthly at 600 S. Michigan Ave., Chicago 5, Ill., by the American Veterinary Medical Association. Second class postage paid at Chicago, Ill., and at additional mailing offices.

PITMAN-MOORE COMPANY DIVISION
ALLIED LABORATORIES, INC.
INDIANAPOLIS

Correspondence

October 9, 1958

Dear Sir:

Having learned from the August issue of the JOURNAL of the AVMA, of which I have been a subscriber for over 30 years, of the death of Dr. Nelson S. Mayo, a fellow practitioner with whom I had a close friendship, I have published the enclosed article, dedicated to his memory, in the *Cuba Ganadera* magazine.

I would really appreciate a reproduction of this article (following) in the JOURNAL.

Thanking you in advance, I remain

Cordially yours,
s/BERNARDO J. CRESPO
Havana, Cuba

American veterinary medicine has lost, through the death of Dr. Nelson S. Mayo, one of the most outstanding representatives it has had. A distinguished professor who possessed an amazing medical culture, an exceptionally enlightened pathologist, and a prodigious writer, his work during the last 50 years has always represented a contribution of unquestionable merit and undisputed value to veterinary medicine in his own country and elsewhere.

During his long sojourn in Cuba, Dr. Mayo held such important posts as Assistant Director General of the Agronomic Experimental Station, and Head of the Animal Industry Department of the same institution, when he devoted a great deal of his time, knowledge, and experience to the study of several diseases that were causing much harm to the Cuban cattle industry. This devotion was rewarded by his discovery of the origin of the diseases in question and of the prophylactic and curative treatments of many of them, the success of which is known to everybody; the Cuban cattle sanitation being indebted to those studies and research for a great part of its present advancement.

Years ago, the University of Havana, acting on the recommendation of its Faculty of Veterinary Medicine, conferred upon him a Doctor *Honoris Causa* diploma, thereby justly recognizing his manifold professional merits and the unusually valuable services rendered by him in the field of veterinary medicine during the time he spent in Cuba.

Dr. Mayo was a close friend of the author of these heartfelt lines. His demise at the age of 91 in his residence of Highland Park, Ill., has been the source of deep sorrow for all of us who knew and loved him. (Sept. 23, 1958)

• • •

September 8, 1958

Dear Sir:

Because of possible radiation hazards, the writer is impelled to comment on a recent JOURNAL article, "Use of Dental Films in Veterinary Practice," (Aug. 15, 1958: 201).

The technique which is described for "radiographing small areas of pet animals" lends itself to dangerous overexposure of any person holding the animal under the cone pictured in figure 5, and offers little advantage over standard techniques. The person in this picture is exposing himself unnecessarily to direct gonadal irradiation. There is evidence that such irradiation, in addition to that which man gets from natural background and required radiation for medical diagnostic purposes, is a potential cause of hereditary damage to future generations. This potential damage is considerably multiplied if a staff member is exposed several times a week.

The authors also recommend the use of dental film packs with a paper clip as an indication of radiation exposure. This method is quite archaic, is of little value because of energy variability, and has been replaced by calibrated film badges.

The use of radiological techniques should be expanded in veterinary medicine, but only if the proper precautions are taken to prevent excessive exposure to the persons involved. The machines used in many practices are obsolete or are operated by individuals who have little knowledge concerning their proper use.

In general, protective devices, such as lead rubber aprons and gloves, the use of fast x-ray film, the use of cones and diaphragms to narrow the x-ray beam, filters to screen out rays that do not contribute to the image, and limiting the use of fluoroscopy wherever possible should minimize the amount of radiation exposure.

It is of the utmost importance that all machines, especially those which are more than ten years old, be checked by a competent radiation physicist. To better understand the use of x-ray machines, recent standard textbooks of radiology should be consulted. In addition, small, informal discussion groups with a local radiologist or radiation physicist have proved to be of immeasurable value.

Radiological techniques are an indispensable part of the veterinarian's armamentarium but they should be used with intelligence and discretion.

Very truly yours,
s/S. MICHAELSON, Rochester, N. Y.

Dr. Wolff Joins Diamond Research Staff

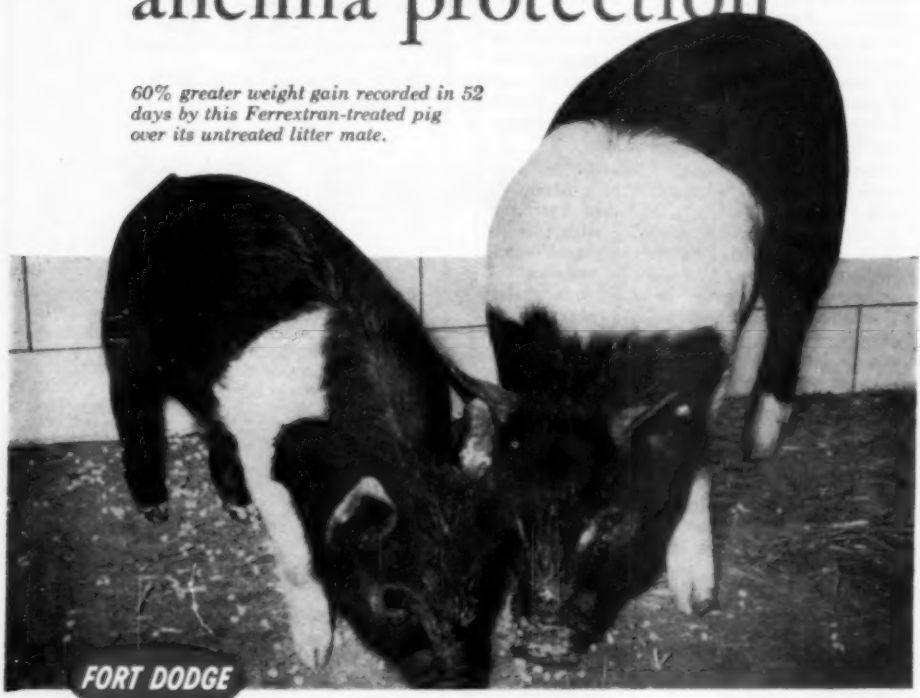
Diamond Laboratories announced recently that Dr. Allen Wolff has joined their biological division as a research associate.

Dr. Wolff received his B.S. degree from Michigan State College in 1953 and his D.V.M. degree from Ohio State University in 1957. He has done a considerable amount of research work which has led to the publication of several scientific papers.

Before coming to Diamond Laboratories, Dr. Wolff was engaged in general practice in Alma, Mich.

This injectable iron
gives persistent, prolonged
anemia protection

60% greater weight gain recorded in 52 days by this Ferrextran-treated pig over its untreated litter mate.



Ferrextran

IRON POLYSACCHARIDE COMPLEX

Ferrextran supplies needed iron, complexed with low molecular weight dextran derivatives, in a form that can be injected without toxic effects, at 2 to 5 days. One injection of a 2 cc. dosage at this age not only overcomes anemia in its earliest stages but sustains hemoglobin levels throughout the critical first 3 weeks to prevent anemia recurrence. All tests show Ferrextran scores highest for persistent, prolonged protection. Packages of 12/20 cc. and 12/50 cc. vials.

FORT DODGE LABORATORIES, INC., FORT DODGE, IOWA

Instructions to Authors

JOURNAL of the AVMA

Exclusive Publication.—Articles submitted for publication are accepted with the understanding that they are not submitted to other journals, which is ethical publication procedure.

Manuscripts.—Manuscripts, including **footnotes, references, and tables,** must be typewritten, **double-spaced,** on 8½- by 11-in. bond paper, and the original and one carbon copy, submitted. One-inch margins should be allowed on the sides, with 2 in. at top and bottom. Articles should be concise. Short, simple sentences are clearer and more forceful than long, complex ones.

Illustrations.—Photographs should be furnished in glossy prints, and of a size that will fit into the JOURNAL of the American Veterinary Medical Association with a minimum of reduction. Photomicrographs which can not be reduced should be marked for cropping to 1-column or 2-column width. Identifying marks within the photomicrographs, such as arrows, letters, or numbers, should be clearly marked with black India ink or white opaque ink to insure good contrast for reproduction and must be large enough to stand reduction, if necessary.

Drawings, graphs, and charts should be made clearly and accurately in India ink on white paper and a glossy print of them submitted when possible. Numbers or letters appearing on graphs or charts should be large enough to allow for any reduction necessary for the chart or graph to fit JOURNAL pages. Blue lines in graph paper drop out in reproduction; therefore, if lines are required they must be drawn in black ink. All illustrations should bear the name of the author and the illustration number on the back.

Tables.—Tables should be simple and typed **double space.** Complex tables are not conducive to perusal. It is wiser to summarize complex material than to tabulate it.

References.—References should be typed **double space,** in alphabetical order, and should be prepared in the following style: name of author, title of article, name of periodical with volume, year, and page numbers. References to journals not commonly known should give the complete name of the periodical, and where published so that they may be added to our reference files. When books are cited, the name of publisher, location, edition, and year should be given.

American Veterinary Medical Association
600 S. Michigan Avenue
Chicago 5, Illinois

CONTENTS—*Continued*

CURRENT LITERATURE

Abstracts

Books and Reports

THE NEWS

MISCELLANEOUS

to treat bovine ketosis, canine dermatoses, inflammatory joint conditions with a duration of action that's "just right"

Ultracortenol®

(prednisolone trimethylacetate CIBA)

ULTRACORTENOL is a new and improved glucocorticoid with distinct advantages over prednisone and prednisolone: the duration of therapeutic action is "just right" and therefore optimally effective. Hence, a single intramuscular injection generally achieves the desired effects, and daily injections or supportive oral therapy are not needed to maintain effective corticoid levels.

ULTRACORTENOL has been extensively tested and enthusiastically accepted by a number of small- and large-animal practitioners[1-10] who found these regimens to be highly effective:

Dairy Cow	Ketosis (acetonemia)	Single 100- to 200-mg. injection*
	Shock ("downer" cow) syndrome	Single 200-mg. injection as supportive therapy.
Dog	Dermatoses	5 mg./10 pounds body weight, total single dose not to exceed 20 mg. For sustained therapy, repeat once or twice a week as indicated.
	Inflammatory joint conditions	5 mg./10 pounds body weight, total single dose not to exceed 20 mg. Supportive oral therapy not necessary.

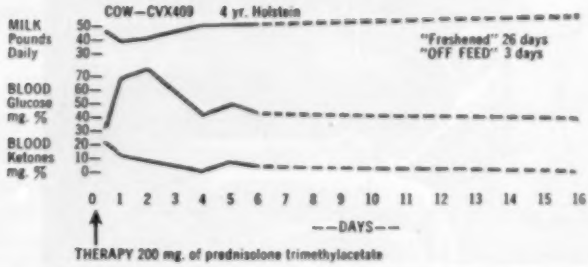
*This initial injection may be reduced to 50 to 100 mg. intramuscularly if simultaneous administration of intravenous glucose is given, thus permitting more economical glucocorticoid therapy. If necessary, either regimen may be augmented by an additional injection of 50 to 100 mg. Ultracortenol after 24 to 48 hours.

C I B A **Effective Veterinary Drugs, Products of Exacting Research**

New injectable glucocorticoid

BOVINE KETOSIS Following a single intramuscular injection of Ultracortenol, investigators observe that:

- appetite is restored, depression is dispelled within 12 to 24 hours[1]
- blood glucose levels are raised within 12 hours[1]
- blood ketone levels return to normal within 24 to 96 hours[1]
- a steady increase in milk production begins after 48 hours.[2]



This chart shows the good response in ketotic cow following intramuscular injection of 200 mg. of Ultracortenol.

(Adapted from Vigue[1])

CANINE DERMATOSES Reporting on 9 animals (of whom 6 received 15 or 20 mg. Ultracortenol in a **single** intramuscular dose), Pollock[3] says, ". . . Ultracorten Trimethylacetate [Ultracortenol] proved effective not only against the seemingly innocuous lesions, but also against the hemorrhagic dermatitis associated with exquisite pain." And, "The duration of the anti-inflammatory phase varies from seven to ten days depending upon the dosage. . . ."[3]

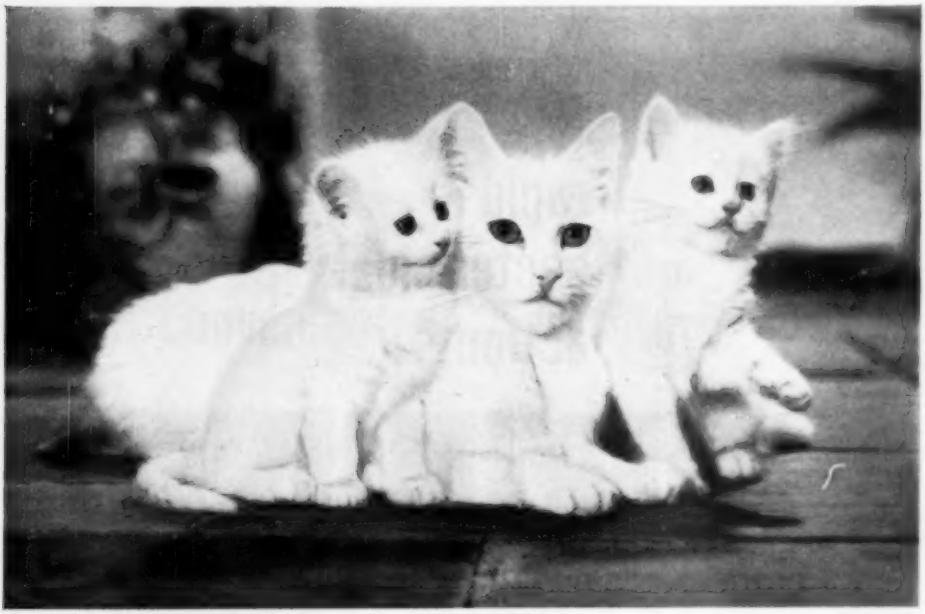
References: 1. Vigue, R. F.: J.A.V.M.A. 133:326 (Sept. 15) 1958. 2. Shaw, J. C.: Personal communication. 3. Pollock, S.: To be published. 4. Rabin, P. H.: Personal communication. 5. Hoffer, S. H.: Personal communication. 6. Weir, H. T., and Hazelrig, J. W.: Personal communication. 7. Beck, J. W.: Personal communication. 8. Bull, W. S.: Personal communication. 9. Fessenden, P. E.: Personal communication. 10. Lohmeyer, C.: Personal communication.

SUPPLIED: Multiple-dose Vials, 10 ml., each ml. containing 10 mg. or 25 mg. of prednisolone trimethylacetate in suspension for injection.

ULTRACORTENOL is available from ethical veterinary distributors throughout the United States.

2/2602 VK

CIBA SUMMIT, NEW JERSEY



Cats quickly respond to Puss 'n Boots

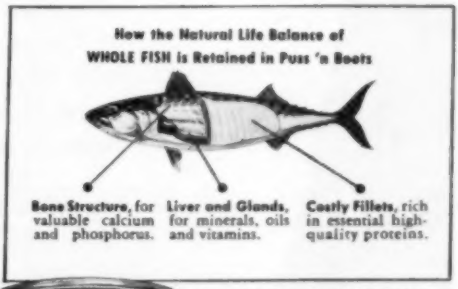
in energy . . . disposition . . . good appearance

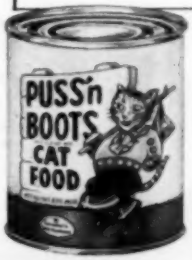
A balanced whole-fish formula means livelier, lovelier pets.

Your cat-owner clientele will be delighted at the improvement in their pets' spirit and appearance when you recommend a regular diet of whole-fish Puss 'n Boots Cat Food. It supplies essential nutrients cats and kittens of all ages are known to need; makes dietary supplements unnecessary.

The natural life balance of whole fish—as the chart shows—is what makes today's Puss 'n Boots so nourishing. Calcium for sound teeth and strong bones, proteins for strength and energy, and vitamins and minerals for all-round health. Selected choice cereals are added for regularity, plus a bonus of vitamins B_1 and E.

It has been the experience of most veterinarians prescribing Puss 'n Boots that the owner sees definite improvement in 3 weeks or less.



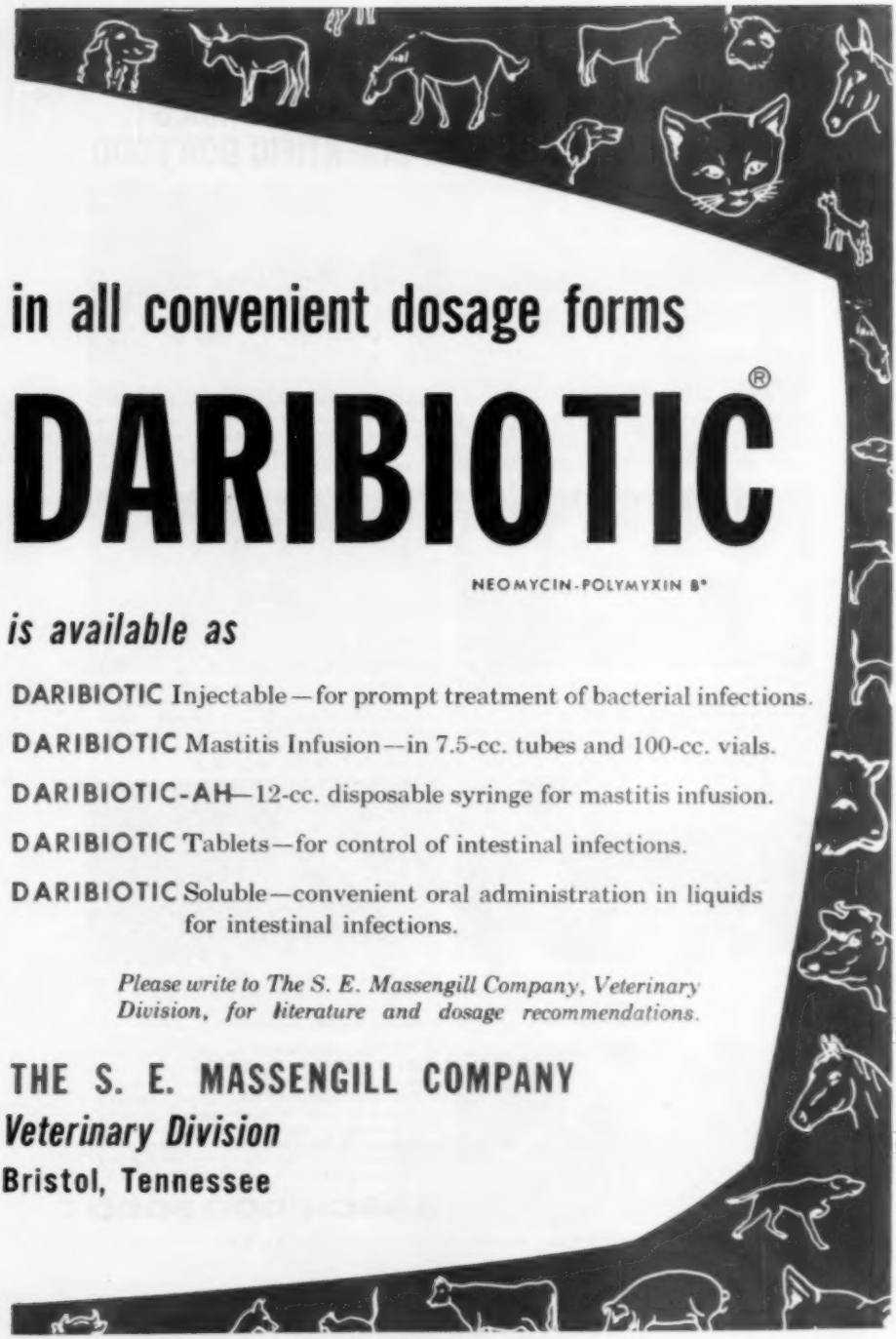


Packed in 8-oz. and 15-oz. sizes

PUSS 'n BOOTS

Fine quality makes it America's largest selling cat food.

Coast Fisheries Division of The Quaker Oats Company, Chicago 54, Illinois.



in all convenient dosage forms

DARIBIOTIC®

NEOMYCIN-POLYMYXIN B*

is available as

DARIBIOTIC Injectable—for prompt treatment of bacterial infections.

DARIBIOTIC Mastitis Infusion—in 7.5-cc. tubes and 100-cc. vials.

DARIBIOTIC-AH—12-cc. disposable syringe for mastitis infusion.

DARIBIOTIC Tablets—for control of intestinal infections.

DARIBIOTIC Soluble—convenient oral administration in liquids for intestinal infections.

Please write to The S. E. Massengill Company, Veterinary Division, for literature and dosage recommendations.

THE S. E. MASSENGILL COMPANY
Veterinary Division
Bristol, Tennessee

BIG REASON WHY KASCO* IS AMERICA'S BEST-RESEARCHED, MOST SCIENTIFIC DOG FOOD

At America's largest dog nutrition research center—the Kasco Research Kennels at Waverly, N. Y.—over 20 years of work has gone into perfecting the balanced, concentrated meal that is today's Kasco Dog Food. This kennel and laboratory, recently expanded at a cost of a quarter-million dollars, has led the way to improved nutrition for the dog and unprecedented economy for the owner.

In these ultra-modern laboratories, trained technicians under Dr. Victor Heiman, Director, have greatly broadened the science of dog nutrition. Their research has enabled them to develop a dog food that is *complete*—for puppies or mature dogs—any size, any breed—even during gestation and lactation.

The 300-dog kennels have advanced lighting and ventilating facilities and a uniquely effective sanitary system. Included are a food-mixing room, kitchen, hospital, metabolism room, maternity ward and isolation ward so Kasco scientists can continue to make Kasco the best dog food you can buy.

Here scientific flavor-preference tests are being made. Each dog is presented with two bowls containing carefully weighed quantities of different dog foods. Every leading dog food has been so tested.

After the dog has eaten its fill, the remaining food is once more weighed to determine which was the more tempting. Through hundreds of such tests, Kasco has retained its superiority.

At Waverly, generations of dogs have been weaned on Kasco and fed this completely balanced food all their lives. Years of tests have proved conclusively that although other foods can be added, Kasco alone provides every nutritive element known to be needed. *It adds up to this: no matter what you are feeding now, switch to Kasco and water. Your dogs will be in better condition than ever before—or will be in just as good condition at a lower cost!*

KASCO* DOG FOOD

VISIT THE FAMED WAVERLY KENNELS AND RESEARCH CENTER AT WAVERLY, N.Y.

AVMA Report

AVMA Activities

President R. E. Rebrassier, Past-President W. W. Armistead, and Dr. J. G. Hardenbergh participated in the Institute of Public Health Practice held at Ann Arbor, Mich., October 7 (*see page 574*).

* * * *

The Judicial Council held its first meeting at headquarters office, October 10. Those attending included: Drs. C. W. Bower, Kansas; F. R. Booth, Indiana, W. G. Brock, Texas; W. O. Kester, Colorado; and Raymond D. Snyder, Pennsylvania. The council elected Drs. Bower and Kingman as chairman and secretary, respectively.

* * * *

The Council on Research met in Chicago October 10 and 11. Those attending the meeting included: Drs. C. A. Brandly, Illinois, C. H. Cunningham, Michigan, L. C. Ferguson, Michigan, Robert Getty, Iowa, W. A. Hagan, New York, Rue Jensen, Colorado, T. Carl Jones, Massachusetts, Hadleigh Marsh, Montana, R. D. Turk, Texas, and J. D. Wheat, California. Drs. Getty and Cunningham were re-elected as the council's chairman and secretary, respectively.

* * * *

The Board of Governors (Drs. D. J. Anderson, R. E. Rebrassier, and S. F. Scheidy) met in the AVMA offices, October 21 and 22, with members of the headquarters staff.

* * * *

The Council on Public Health and Regulatory Veterinary Medicine held its first meeting at the AVMA offices on October 23. Members of the council attending the meeting included: Drs. R. K. Anderson, Minnesota, John Cunkelman, Illinois, J. R. Hay, Illinois, S. L. Hendricks, Iowa, Oscar Sussman, New Jersey, E. S. Tierkel, Georgia, C. D. Van Houweling, District of Columbia, Asa Winter, Michigan, and E. W. Young, District of Columbia. The council elected Dr. Geyer and Dr. Hendricks as chairman and secretary, respectively. The council's next meeting will be held March 9 and 10, 1959.

* * * *

Section officers (Committee on Program) met at Association headquarters on October 24 with members of the staff, to discuss and plan the scientific program for the Joint Pan American-AVMA meeting to be held in Kansas City, Mo., Aug. 24-27, 1959. Section officers (*see* JOURNAL, *Nov. 1, 1958, adv. p. 28*) attending the meeting were: Drs. E. F. Ebert, Missouri; John S. Haley, M. J. Twiehaus, and F. H. Oberst, Kansas; D. K. Detweiler, Pennsylvania; C. Edwin Hofmann and Wm. F. Irwin, Oklahoma; M. S. Cover, Delaware; S. B. Hitchner, Wisconsin; S. J. Roberts, New York; and F. A. Todd, District of Columbia.

Mr. Walter Lawrence, R.C.A., Mr. Wallace S. Sims, Pitman-Moore Co., and Dr. L. E. Fisher, TV coordinator, met with the section officers to assist in planning the closed-circuit TV portion of the program. Dr. Juan Figueroa, co-chairman of the Organizing Committee also participated in this meeting.

* * * *

Election ballots were mailed October 31 for Executive Board Districts II, VII, and IX. Polls will close Dec. 1, 1958.

(*Continued on adv. p. 21*)

Are we playing with DYNAMITE?

Livestock efficiency has reached new peaks through the combined efforts of research scientists, suppliers, veterinarians and farmers. Together, we've made great progress in recent years.

Yet today more and more farmers are doing their own veterinary work. Aiding them are suppliers who sell for non-professional use. Both farmers and suppliers have every legal right to do this. But we wonder where such practices will end.

Are they in the best interests of the livestock industry? Or, are we playing with dynamite?

You can help . . . protect the future of the livestock industry and your own profession by taking a firm stand on this one big issue: Whether or not to buy from a supplier who also sells direct to laymen.

Why not buy from an ethical house, one that sells only to graduate veterinarians? We are on your team *all* the time, not just when it's economically expedient!

AFFILIATED LABORATORIES CORPORATION

WHITE HALL, ILLINOIS

Corn Belt Laboratories, Inc. — The Gregory Laboratory, Inc.

The National Laboratories Corp. — Grain Belt Supply Co.

Affiliated

Produced for and sold to Graduate Veterinarians only

THE KEN-L-BISKIT DOG:

healthier... handsomer... stands out from the rest

Costs only PENNIES A DAY to feed him the Dog Food of Champions

Ken-L-Biskit . . . so nutritious, it's the official food at more A.K.C. shows than all other dog foods combined. So dependable, America's top kennels have used it to develop generation after generation of champions. So digestible, even puppies thrive on it. Complete . . . needs no expensive supplements. Get it today, in the thrifty fifty-pound bag.

AVMA REPORT — continued —

AVMA President Rebrassier participated in the Southern V.M.A. meeting held in Memphis, Tenn., October 26-30.

★ ★ ★ ★

Meeting of delegates and alternates to the House, and secretaries of constituent associations in Executive Board Districts III, VII, and VIII, met Sunday, October 26, in Memphis, Tenn., prior to the meeting of the Southern VMA. Those participating in the program included President Rebrassier, Executive Board member McKenzie Heath, and members of the AVMA staff (Executive Secretary Kingman, Associate Editor Price, J. A. McCallam, Washington office, and Brian Forster, Public Information).

★ ★ ★ ★

Dr. J. G. Hardenbergh and Mr. Russell Rongren met with the Committee on Local Arrangements on October 30 in Kansas City to discuss plans for the Joint Pan American-AVMA convention to be held in that city.

★ ★ ★ ★

Executive Secretary Kingman and Editor Aitken participated in the National Swine Industry Conference, held at Purdue University, November 3 and 4.

★ ★ ★ ★

President-elect S. F. Scheidy and Dr. Donald A. Price, represented the AVMA at the meeting of U.S.L.S.A. held in Miami Beach, Fla., November 4-7.

a true therapeutic restraint

Trilafon* veterinary

(pronounced Trill'-ah-fon) perphenazine

- controls the intractable animal without physical force
- achieves the desired degree of tranquilization without dulling normal responses

safe, effective dosage range for both large and small animals

animal	oral	intravenous	intramuscular
 DOG	4 mg./10 lb. b. i. d.	5 mg./20 lb.	5 mg./20 lb.
 SWINE		10 mg./100 lb.	10 mg./100 lb.
 ADULT BOVINE		75-125 mg.	100-150 mg.
 CALVES & HEIFERS		adjust adult dosage according to body weight and degree of tranquilization desired.	

NOTE: Refer to Schering literature for complete information regarding dosage, side effects, precautions and contraindications.

PACKAGING: TRILAFON Tablets, 4 and 8 mg., bottles of 50 and 500.

TRILAFON Injection, 5 mg./cc., 10 cc. multiple-dose vial, *Small Animals* boxes of 1 and 6;
25 mg./cc., 30 cc. multiple-dose vial, *Large Animals* boxes of 1 and 6.

Schering

*T.M. V-TR-J-297

VpC VIA-D-MINERAL SUPPLEMENT

— your client's key to more profit per cow

Here's proof that today's pasture and grains can't build and maintain a high-profit dairy herd by themselves: *The average U.S. dairy cow produces only* 209 *pounds of butterfat each year.* Today's pasture and grains need help—exactly the kind of help Via-D-Mineral Supplement is formulated to supply. Via-D-Mineral provides 100,000 USP units of Vitamin D_2 per pound—plus Vitamins A and B-complex, calcium, phosphorus, iodine, iron, manganese, copper, potassium, zinc, cobalt and active dry yeast. Via-D-Mineral is important in building resistance to disease . . . preventing nutritional disorders . . . boosting milk and fat production. Remind your clients that one cow producing 500 pounds of fat annually will make as much net profit for them as *three* cows producing only 250 pounds apiece. The key to this profit can be Via-D-Mineral fed before breeding, during gestation, after parturition—to every animal in the herd, every day.

VpC Supplements Nationally Advertised To Your Clients!

Farmers in your area are reading about the cash-saving, production-increasing values of Vitamineral Supplements in FARM JOURNAL, FARM QUARTERLY and other farm papers. Each VpC advertisement emphasizes that only you, as a veterinarian, are authorized and qualified to recommend and dispense Vitamineral Supplements.

31st Ed. of VpC Feed Book now ready. Send for your supply.

VITAMINERAL PRODUCTS CO.,

PEORIA, ILLINOIS

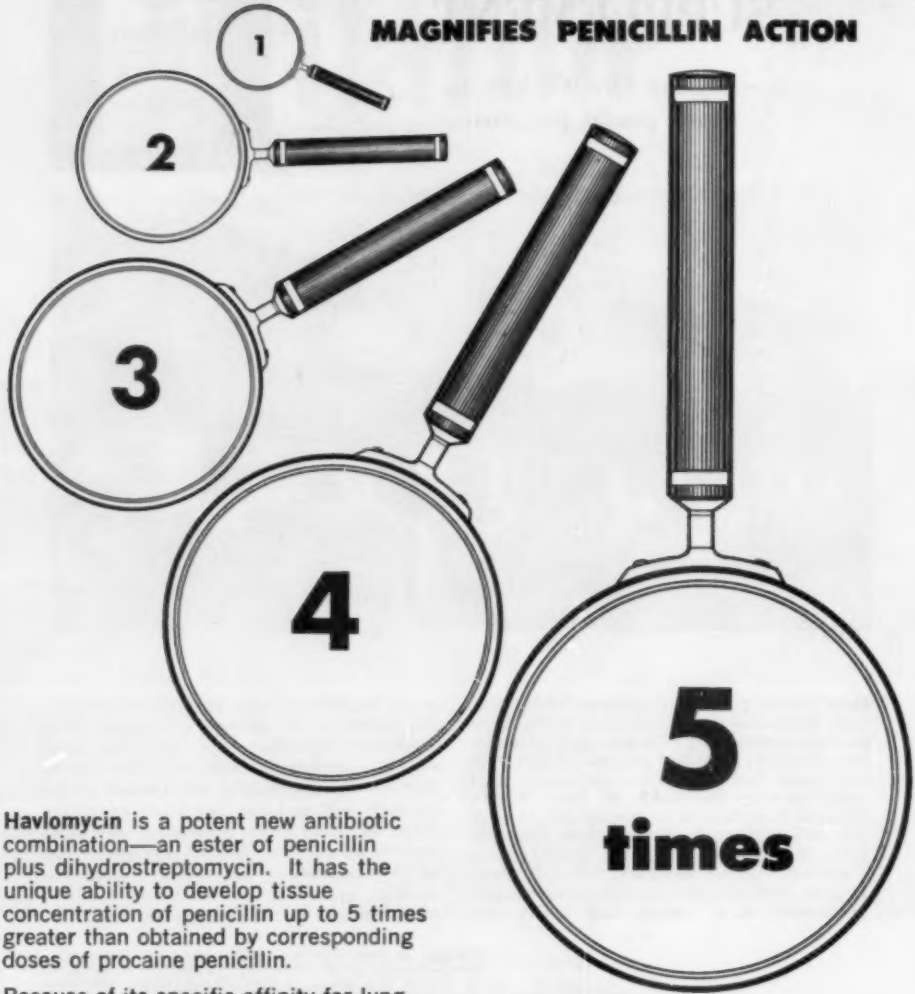
WRITE FOR CONFIDENTIAL PRICE LIST ON:

Via-D-Mineral . . . Con-o-mineral . . . Viamineral . . . Viaferm . . . Ribad . . . "A to Z" . . . VpC Dog Food Supplement

NEW

HAVLOMYCIN

MAGNIFIES PENICILLIN ACTION



Havlomycin is a potent new antibiotic combination—an ester of penicillin plus dihydrostreptomycin. It has the unique ability to develop tissue concentration of penicillin up to 5 times greater than obtained by corresponding doses of procaine penicillin.

Because of its specific affinity for lung and mammary tissues, Havlomycin is quick and effective for respiratory infections in large and small animals and for **intramuscular** mastitis therapy.

Dosage: large animals, 1 cc. per 100 lb. body weight once daily for 2-4 days.
small animals, 0.5 cc. to 1.5 cc. once daily for 2-4 days.

Supply: 10 cc. vial with vial of sterile diluent.

HAVER-LOCKHART
LABORATORIES

Kansas City, Missouri

equipment news!

precision veterinary equipment

NICHOLSON

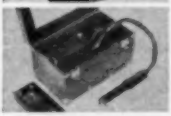
FRANK'S FETAL EXTRACTOR helps you through difficult OB cases. Portable, quick and easy-to-assemble. Write for folder.

PLEXI-SIGN LETTERS in all sizes and colors, install them with a screwdriver or adhesive cement. Write for folder.

PLASTI-PLATED CAGE WARDS are simple to put together. We send you materials, full instructions. Write for folder.

RADIANT WARMING PANEL is plasti-plated to resist clawing, stains, urine. Shock-proof. Write for folder.

INSTRUMENT AND SERUM CASE has baked black enamel over steel. Brass trim. Six models. Write for folder.



ELECTRIC FIRING IRON. the modern way to fire horses, remove growths, tumors. Write for folder.

ELECTRIC BRANDING IRON heats in 90 seconds, doesn't smoke, weighs 11 ounces. B, T, V brands. Write for folder.

NEW COPON KENNEL PAINT gives hard, baked-type finish. You can brush, roll or spray it on. Write for folder.

NEW TRANS-JECTOR electronic ejaculator weighs only 16½ pounds, has no fragile vacuum tubes. Write for folder.

SWANSON VACCOMATIC SYRINGE will help you vaccinate up to 400 head of cattle an hour. Write for folder.

NEW IncinaMATOR is first really fool-proof veterinary clinic incinerator and cremator. Write for folder.

Your veterinary hospital can have a "New Look" with our Plexi-Sign letters

Our new plastic letters make your veterinary hospital sign professional and modern as it should be. These durable Plexi-Sign letters have a clean, modern look, and they won't rust or fade.

You can make your own sign by ordering individual letters from Nicholson. They're easy to install with just a screwdriver or adhesive cement. Or you can also order complete ready-made signs if you wish. Mail the coupon today for detailed information on sizes and prices. Send sketch of sign you want for quotation on do-it-yourself or ready-mades at no obligation.

- ☐ fetal extractor
- ☐ Plexi-Sign letters
- ☐ Plasti-Plate
- ☐ warming panel
- ☐ instrument serum cases
- ☐ firing iron
- ☐ branding iron
- ☐ Copon paint
- ☐ Trans-Jector ejaculator
- ☐ Swanson syringe
- ☐ IncinaMATOR disposer

Please send me free folders on the items I've checked. No cost or obligation, of course.

DVM

street

city

Nicholson Manufacturing Inc.

3990 Ulster St., Denver 7, Colorado

clip and mail today

Effective treatment of
TAPEWORM INFESTATION

- **Vermifugal and purgative in action, NEMURAL is particularly effective in dogs and adult cats. Moreover, *it acts quickly.***
- May be administered without a preliminary starvation. Administration usually not followed by emesis.
- Definite dosage: One 18 mg. tablet for each 8 lb. of body weight.

Literature and detailed information supplied on request.

Supplied in tablets of 18 mg., bottles of 100 and 500. (Each tablet contains 3.13 mg. arsenic.)

Winthrop LABORATORIES NEW YORK 18, N. Y.

Nemural (brand of drocarbil), trademark reg. U.S. Pat. Off.

NEMURAL®

for **MASTITIS** *of*

dry and lactating cows

NEW SQUEEJET T.M.*

single-dose dispenser for

FURACIN® **SOLUTION** *veterinary*

brand of nitrofurazone

- delivers one full dose
- is discarded after using
- saves time
- is convenient, especially when a number of cows are to be treated
- unique package

*design patented

FURACIN has been shown to be strikingly effective in controlling mastitis under field conditions.[1,2] Of 7,123 lactating cows with acute mastitis, fair to excellent results were obtained in 5,597 (78%). Of 3,418 dry cows which had had mastitis during their previous lactating period, fair to excellent results were obtained in 3,104 (90%).

SUPPLIED A water-miscible liquid of FURACIN 0.2% in polyethylene glycol and water. Single-dose SQUEEJET disposable dispenser of 30 cc. Still available, bottles of 500 cc. with rubber stopper and 1 gal.

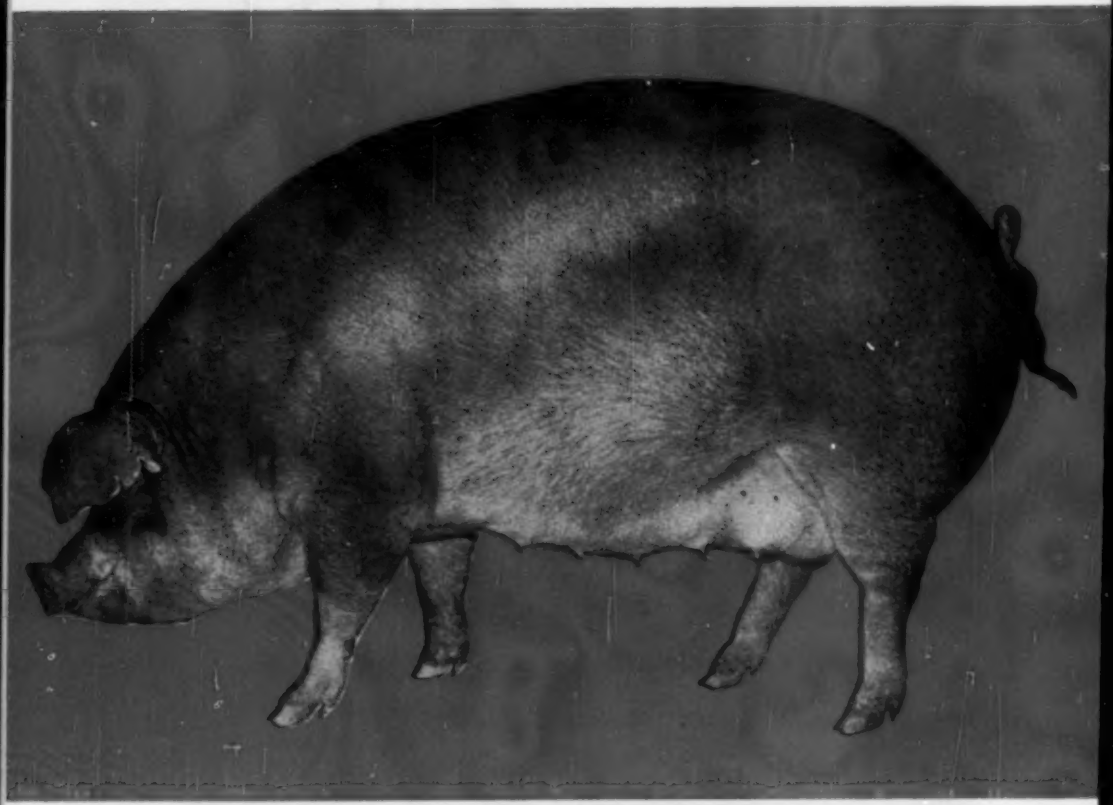
1. Mires, M. H., and Chadwick, R. H.: Vet. News **10**:3 (Jan.-Feb.) 1947. 2. Mires, M. H.: J. Am. Vet. M. Ass. **117**:49 (July) 1950.

All Eaton Veterinary Specialties Are Available Through Your Professional Veterinary Distributor.

NITROFURANS—a *new* class of antimicrobials— neither antibiotics nor sulfonamides

EATON LABORATORIES, NORWICH, NEW YORK

THE DUROC JERSEY is believed to be descended from various breeds of red hogs imported from Europe as early as 1804. It was not until 1880, however, that the modern Duroc was developed. The breed combines large size with feeding capacity and good ability to reproduce.



"M·L·V", EXCLUSIVE TRADEMARK of Fort Dodge Laboratories, designates Fort Dodge Brand Modified Live Virus Hog Cholera Vaccine. M·L·V is the original, first-licensed such vaccine. It is prepared exclusively from swine tissues from pigs inoculated with virus previously modified in virulence by serial passage in rabbits. Solid immunity of more than three years has been demonstrated.

FORT DODGE®

M·L·V TRADEMARK

The original modified live virus vaccine.

U. S. Patent RE-23746

FORT DODGE LABORATORIES, INC., FORT DODGE, IOWA

JOURNAL
of the American Veterinary Medical Association
Established January, 1877 Chicago 5, Illinois
VOL. 133 December 1, 1958 No. 11

Some Metabolic Derangements Associated with Magnesium Metabolism in Cattle

ROBERT R. MARSHAK, D.V.M.

Philadelphia, Pennsylvania

AS EARLY as 1929, a relationship was suggested[23] between hypomagnesemia and the occurrence of tetany in dairy cattle newly turned out on spring grass. European and American workers confirmed these observations and described a similar syndrome in beef cattle on winter rations.[4,16-18,21,22,24,25,27]

This paper questions a widely accepted hypothesis that the magnesium ion plays more than an incidental role in the etiology of tetany in adult cattle as it occurs in the United States. The primary disturbance appears to be hypocalcemia which, although widely reported, is often minimized.

THE ROLE OF HYPOMAGNESEMIA

If "pure" hypomagnesemic tetany occurs in adult cattle, it has seldom been proved. On the other hand, there can be little doubt that plasma magnesium may fall to very low levels in the absence of clinical signs.[1,2,4,6,22]

Seasonal hypomagnesemia has been reported by numerous workers.[1,2,6,16,17] The significance of this fluctuation is not clear and must await better understanding of the fundamentals of magnesium metabolism. Some experimental evidence indicates that seasonal hypomagnesemia may be related to high ruminal ammonia production reported for cattle on spring grass.[14,15]

There is speculation that this or unknown dietary factors may block the availability of the magnesium abundantly present in young grass. The mechanisms are unknown. The possibilty exists that chelating agents, such as phytin, may be present in high concentration in young herbage, binding magnesium and other dietary cations into insoluble complexes.

Attempts to correlate hypomagnesemia with the occurrence of inclement climatic conditions have not been convincing.[1,3,16,17] In this regard, the author cautiously acknowledges a distinct impression that many of his "grass tetany" cases were attended in cold rainy weather, or at least in fields still moist with recent rain.

The fact that the seasonal incidence of hypomagnesemia and tetany may coincide does not prove a cause and effect relationship. Such a relationship is questionable in view of the common occurrence of asymptomatic hypomagnesemia as well as a non-hypomagnesemic form of the syndrome. The constant finding of hypocalcemia, and the known clinical effect of plasma calcium deficit, can not be minimized.[11,28] Moreover, the efficacy of unfortified calcium salt therapy is widely recognized.[11,28]

The heavy use of fertilizers, particularly potash and nitrogen, has long been suspected of lowering blood magnesium levels of cattle grazing on treated pastures.[4,5,17,21] The evidence for this is not substantial[3] and the complexity of ionic interrelationships should discourage formulation of "comfortable" suppositions.

Based on present knowledge, the clinical significance of hypomagnesemia in cattle can not be properly assessed. The importance of magnesium in the body economy is beyond question.[5,6,9,10,12,13] It is a major intracellular cation, functioning as a metallic activator of enzyme systems, and is vitally concerned with neuromuscular conduction.

The high incidence of asymptomatic hypomagnesemia would seem to indicate that levels of plasma magnesium do not necessarily reflect levels of cellular magnesium.

From the School of Veterinary Medicine, University of Pennsylvania, Philadelphia.

The author thanks Drs. Robert Kenney, W. B. Boucher, and J. E. Croshaw for procuring cows with grass tetany for this study.

Presented before the Section on General Practice, Ninety-Fifth Annual Meeting, American Veterinary Medical Association, Philadelphia, Pa., Aug. 18-21, 1958.

Even in severe magnesium deficiency states, the soft tissues are able to maintain a remarkably constant magnesium content at the expense of large, readily mobilizable skeletal depots.[9,19,26] As with phosphorus, plasma magnesium may fluctuate widely without apparent clinical effect.

In the United States, nonparturient tetany appears typically as a hypocalcemia of multiparous cows. In dairy animals, tetany usually occurs in high-producing, pregnant cows recently turned out on spring grass;[11,26] beef cattle are more commonly affected on winter pastures in the early postpartal period.[15,27]

Plasma magnesium levels are variable within a wide range. In any particular herd, hypomagnesemia is noted equally in healthy animals and animals with hypocalcemic tetany.[27] There is little reason to suppose that its clinical impact is more than coincidental. A common omission in studies of tetany is failure to sample normal control animals in the herd under surveillance.

BIOCHEMICAL STUDIES

Pretreatment chemical analyses on serums from cows with tetany in eight herds are given (table 1). They illustrate consistent hypocalcemia with marked variation in serum magnesium. In this series, serum calcium levels ranged from 4.5 to 7.2 mg. per 100 cc.; serum magnesium from 0.61 to 3.9 mg. per 100 cc. Blood values from 2 cows with parturient paresis are inserted for purposes of comparison. All animals showed marked clinical signs ranging from severe tetany to coma.

Except for extreme variability in serum magnesium, the biochemical picture in the peripheral blood of dairy cows with tetany and parturient paresis is remarkably similar. Phosphorus tends to move with calcium; blood sugar is elevated; sodium and potassium are within normal limits.

The important question of acid-base balance was not investigated.

While it appears unlikely that hypomagnesemia causes tetany in adult cattle, the level of plasma magnesium may influence the clinical configuration of the hypocalcemic seizure. Particular importance has been attached to the calcium-magnesium ratio.[7,18,28] The minimal tetany and tendency toward coma in parturient paresis is attributed to a low calcium-magnesium quotient.

Conversely, the hyperirritability, violent tetany, and convulsions commonly observed in animals with grass tetany are ascribed to a high calcium-magnesium quotient. Thus, one might expect cow F (Ca-Mg quotient=5) to show less tetany than cow B (Ca-Mg quotient=10) or cow E (Ca-Mg quotient=7) (table 1). The fact is that cow F showed violent tetany, whereas cows B and E appeared clinically to have parturient paresis. It seems evident that the intensity of tetany is more than a simple function of the calcium-magnesium ratio.

DISCUSSION

The basis for hypocalcemia not immediately associated with the onset of lactation in cattle is poorly understood. As in parturient paresis, it appears unrelated to

TABLE 1—Results of Pretreatment Chemical Analyses on Serums from Cows with Tetany in Eight Herds*

Cow	Breed	Age (yr.)	Ca (mg./100 cc. blood)	Mg (mg./100 cc. blood)	Ca-Mg quotient	P (mg./100 cc. blood)	Na (mEq/L)	K (mEq/L)	Sugar (mg./100 cc. blood)	NPN (mg./100 cc. blood)	Interval since last parturition (mo.)	Months pregnant
A	G	9	4.5	3.9	1.2	3.0	140.0	4.0	128.0	30.0	7	4
B	G	8	6.2	0.61	10.0	2.8	145.0	5.4	.....	.....	1	..
C	G	8	6.0	2.4	2.5	2.6	144.0	4.0	102.9	20.3	8	5
D	J	8	7.0	1.3	5.4	2.9	154.0	3.8	81.3	50.1	8	4
E	J	6	7.2	1.01	7.0	2.2	150.0	2.9	84.0	23.1	1	..
F	G	Aged	5.0	1.01	5.0	1.7	.....	.....	.....	.....	4	2
G	G	6	6.0	3.3	1.8	.....	138.0	2.3	.....	.....	5	3
H	J	Aged	5.6	3.2	1.7	2.5	131.0	4.1	.....	.....	..	..
H-4 (Parturient paresis)	J	6	5.3	4.1	1.3	3.0	140.0	3.6	96.0	26.0	..	..
E-100 (Parturient paresis)	J	7	4.4	2.9	1.5	1.7	137.0	3.9	86.0	26.0	..	..

*Methods of chemical analyses were based on the following: Calcium—Fiske, S. H., and Logan, M. A.: J. Biol. Chem., 93, (1931): 211. Magnesium—Orange, M., and Rhein, H. C.: J. Biol. Chem., 189, (1951): 379. Phosphorus—Fiske, S. H., and Subbarrow, Y.: J. Biol. Chem., 66, (1925): 375. Sugar—Folin, O., and Wu, H.: J. Biol. Chem., 41, (1920): 367. NPN—Folin, O., and Wu, H.: J. Biol. Chem., 38, (1919): 81. Sodium and potassium: flame photometry.

dietary calcium deficiency and generally responds well to simple intravenous calcium salt therapy. It is significant that morbidity is largely limited to multiparous females in the advanced age group. Beef cows wintering in the open prior to the appearance of new spring pasture usually sicken in the early postnatal period.

In the West, pregnant and parturient beef cows shipped into the area to graze on wheat from fall to early spring are subject to tetany.[20] Debility and poor condition appear to be predisposing factors.

Studies of tetany in American beef cattle have usually disclosed hypocalcemia with hypomagnesemia.[11,18,27,28] In a rare instance, in which normal cows and cows with tetany in a single herd were studied simultaneously, all values for plasma magnesium were well below the accepted normal.[27] Significantly, only animals with hypocalcemia showed clinical signs.

In pondering the pathophysiology of tetany in beef breeds, it is convenient to consider the casual events in parturient paresis of dairy cows. During late pregnancy, the dairy cow which is prone to parturient paresis is in severe negative calcium balance,[29] presumably the aftermath of a rapid series of pregnancies and lactations. At parturition, clinical hypocalcemia is precipitated by the acute calcium deprivation stress of another beginning lactation. Ordinarily, the dairy cow enjoys a high plane of nutrition.

The beef cow experiences the calcium withdrawal stress of multiple pregnancies but her lactation stress is of short duration. Her plane of nutrition under winter conditions is often minimal. In such circumstances, the excessive milk drain by a rapidly growing calf may precipitate hypocalcemia. The clinically recognizable deficit is probably preceded by a relatively long prodromal state of deficit development.

It is noteworthy that tetany in beef cows occurs principally in the early postpartal period when the calf's entire nutrition consists of dam's milk.

In dairy cows, too, the cause of tetany has not been established. Based on the usual circumstances of advanced age, superior lactation performance, and pregnancy, it appears that grass tetany is a parturient paresis-like syndrome elicited by the added lactation stimulus of new succulent pasture.

The age and sex incidence of tetany argue against the concept that nonspecific influences to which the entire herd is equally exposed, such as weather and fertilizing practices, are by themselves responsible for hypocalcemia.The effect of age on calcium and magnesium homeostasis is noteworthy. As a cow grows older, there is less bone calcium and magnesium available for exchange with plasma calcium and magnesium.[5,19]

It is difficult to assess the contributory role of nonspecific environmental factors in disturbing calcium and magnesium homeostasis. In general, the mechanisms regulating the plasma magnesium level are less sensitive than the regulatory mechanisms for plasma calcium. The largely intracellular position of magnesium as compared to calcium would appear not to necessitate such close extracellular compliance.

THERAPY

The therapy of tetany and parturient paresis are identical. Intravenous calcium gluconate and udder insufflation are generally effective. The addition of magnesium salts to calcium solutions appears to offer no advantage over calcium alone. In a limited series of animals treated solely by udder insufflation, there were no relapses.

Veterinarians reporting a higher mortality rate for tetany cases as compared to parturient paresis should consider the longer average time lapse from onset to treatment. This results from the limitations imposed by pasture conditions. The physical stress and possible remote effects of violent tetany, when present, may be another obstacle to recovery.

SUMMARY

A study of 8 adult dairy cows with tetany indicated that hypocalcemia was the primary disturbance. The concept that low plasma magnesium causes tetany is questioned.

References

[1]Allcroft, W. M.: Seasonal Hypomagnesemia of the Bovine without Clinical Symptoms. With Special Reference to the Influence of Climate on the Level of Serum Magnesium and the Incidence of Clinical Hypomagnesemia. Vet. J., *103*, (1947): 75.

[2]Allcroft, W. M., and Green, H. H.: Seasonal Hypomagnesemia of the Bovine without Clinical Symptoms. J. Comp. Path. and Therap., *51*, (1938): 176.

[3]Alten, F., Rosenberger, G., and Welte, E.: Zur

Frage der Ursache und des Wesens der Weidentetanie. Zentralbl. Veterinärmed., *5*, (1958): 201.

[4]Bartlett, S., Brown, B. B., Foot, A. S., Rowland, S. J., Allcroft, R., and Parr, W. H.: The Influence of Fertilizer Treatment of Grassland on the Incidence of Hypomagnesemia in Milking Cows. Brit. Vet. J., *110*, (1954): 3.

[5]Blaxter, K. L., and McGill, R. F.: Magnesium Metabolism in Cattle. Vet. Rev. and Annotations, *2*, (1956): 35.

[6]Bouckaert, J. H., and Oyaert, W.: Some Factors Affecting Magnesium Content of Cow's Blood. Proc. Fifteenth Internat. Vet. Congr. Part 1. Stockholm, *1*, (1953): 571.

[7]Dukes, H. H.: The Physiology of the Domestic Animals. 7th ed. Comstock Publishing Co., Ithaca, N. Y., 1955.

[8]Ducan, G. G.: Diseases of Metabolism. 3rd ed. W. B. Saunders Co., Philadelphia, 1954.

[9]Elkinton, J. R.: The Role of Magnesium in the Body Fluids. Clin. Chem., *3*, (1957): 319.

[10]Elkinton, J. R., and Danowski, T. S.: The Body Fluids. Williams and Wilkins Co., Baltimore, 1955.

[11]Fincher, M. G. (ed.): Diseases of Cattle. American Veterinary Publications, Inc., Evanston, Ill., 1956.

[12]Flink, E. B., McCollister, R., Prasad, A. S., Melby, J. C., and Doe, R. P.: Evidences for Clinical Magnesium Deficiency. Ann. Int. Med., *47*, (1957): 956.

[13]Greenberg, D. M., and Tufts, E. V.: The Nature of Magnesium Deficiency. Am. J. Physiol., *121*, (1938): 416.

[14]Head, M. J., and Rook, J. A. F.: Hypomagnesemia in Dairy Cattle and Its Possible Relationship to Ruminal Ammonia Production. Nature, *176*, (1955): 262.

[15]Head, M. J., and Rook, J. A. F.: Some Effects of Spring Grass on Rumen Digestion and Metabolism of the Dairy Cow. Proc. Nutr. Soc., *16*, (1957): 25.

[16]Inglis, J. S. S., Weipers, M., and Marr, A.: Some Observations on Bovine Hypomagnesemia. Vet. Rec., *66*, (1954): 353.

[17]Kemp, A., and 't Hart, M. L.: Grass Tetany in Grazing Milking Cows. Netherlands J. Agric. Sci., *5*, (1957): 4.

[18]Mershon, M. M., and Custer, F. D.: Tetany in Cattle on Winter Rations. Part 1—A Clinical Report. J.A.V.M.A., *132*, (May 1, 1958): 396.

[19]Neuman, W. F., and Neuman, M. W.: The Chemical Dynamics of Bone Mineral. University of Chicago Press, Chicago, 1958.

[20]Redmond, H. E.: Wheat Poisoning in Cattle. Southwest. Vet., *3*, (1950): 305.

[21]Simesen, M. G.: Hypomagnesaemi—Graestetani. Nord. Vet. Med., *9*, (1957): 305.

[22]Sjollema, B., Grashuis, J., Van Koetsveld, E. E., and Lehr, J. J.: Onderzoekingen over Kopziekte. Tijdschr. Diergeneesk., *80*, (1955): 579.

[23]Sjollema, B., and Seekles, L.: Over de Stoormissen der Minerale Stofwisseling bij Ralfen Ropziekte. Tijdschr. Diergeneesk., *56*, (1929): 979.

[24]Sjollema, B., Van Koetsveld, E. E., Grashuis, J., and Lehr, J. J.: Investigations into Grass Tetany II. Tijdschr. Diergeneesk., *80*, (1955): 1111.

[25]Stewart, J., and Reith, J. W. S.: The Effects of Magnesian Liming on the Magnesium Content of Pasture and the Blood Level of Magnesium in Cows. J. Comp. Path. and Therap., *66*, (1956): 1.

[26]Suter, C., and Klingman, W. O.: Neurologic Disorders Linked to Magnesium Depletion. Neurology, *5*, (1955): 691.

[27]Udall, R. H.: Low Blood Magnesium and Associated Tetany Occurring in Cattle in the Winter. Cornell Vet., *37*, (1947): 314.

[28]Udall, D. H.: The Practice of Veterinary Medicine. 6th ed. Published by the author, Ithaca, N. Y., 1954.

[29]Ward, G. M.: Calcium Balances and Changes of Some Blood and Urinary Constituents as Related to Parturient Paresis in Dairy Cows. Ann. New York Acad. Sci., *64*, (1956): 361.

Magnesium and Tetany Paresis

The tetany which occurs in dairy cows on pasture in Norway is believed to be a conditioned magnesium deficiency rather than (or in addition to) a lack of magnesium in the diet.

Although generous amounts of nitrates in the grass have been mentioned as a conditioning factor, because of the production of large amounts of ammonia in the rumen, the results of feeding experiments do not verify this.

Instead, increasing the sulfur fertilization may be a key factor. This is accomplished when ammonium sulfate, potassium sulfate, or superphosphate are applied. Superphosphate contains more sulfur than phosphorus. The excess of sulfur tends to reduce the rate at which plants take magnesium from the soil. Heavy applications of potassium show the same tendency. The authors suggest the possibility that plants form compounds of nitrogen with sulfur more readily than with magnesium, and hence contain less magnesium following fertilization with sulfates.

Substances present in the grass, such as a high potassium or manganese content, may render magnesium less absorbable or may in some way antagonize or interfere with metabolism by the animal. However, results of tests have not conclusively proved this.

Starvation or underfeeding and low magnesium intake during the stable feeding period just prior to the pasture season seem to predispose to tetany and paresis. Wide deviations from normal numbers and types of rumen microflora occur under these conditions.

(The treatment of tetany and paresis was similar to that used in America.)—*Ender et al. in Nord. Vet.-med., 9, (Dec., 1957): 881.*—R. KLUSSENDORF.

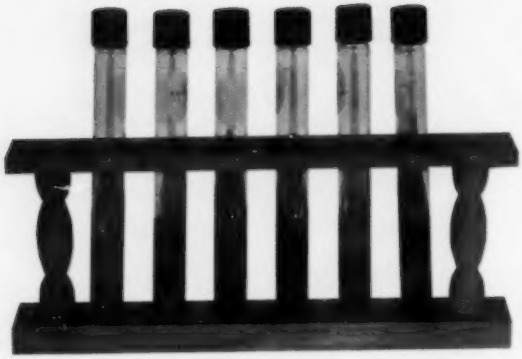


Figure 1

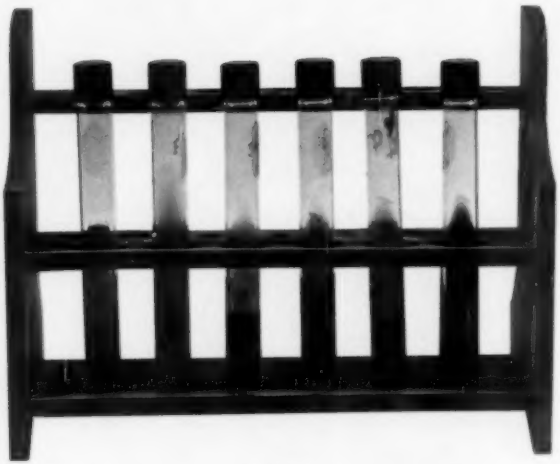


Figure 2

Triple Sugar Iron Agar as an Aid in the Diagnosis of Erysipelas

C. L. VICKERS, D.V.M., and B. W. BIERER, V.M.D.

Columbia, South Carolina

WHILE TRIPLE sugar iron agar is recommended as a medium for use in the identification of Gram-negative enteric pathogens,[1] it has proved of value in the Clemson College diagnostic laboratory in the isolation and identification of *Erysipelothrix rhusiopathiae*. From Dec. 1, 1956, to Dec. 1, 1957, a diagnosis of erysipelas was made in 15 herds of swine and 25 flocks of turkeys, using triple sugar iron agar as an adjunct to the diagnosis.

The ability of an organism to ferment lactose, saccharose, and dextrose with the formation of acid and gas, and its ability to produce hydrogen sulfide, can be determined by the use of triple sugar iron agar as a medium. Hydrogen sulfide is produced by Erysipelothrix, and most strains ferment glucose, lactose, and levulose,[2] but no gas is formed. It is the production of hydrogen sulfide by Erysipelothrix, as it is growing in the triple sugar iron agar medium, that enhances a rapid and accurate diagnosis of erysipelas. The procedure used in this laboratory is simple and would seem to be practical for the practitioner's laboratory.

PROCEDURE

Triple sugar iron agar is purchased in prepared powder form.* It is rehydrated by suspending 65 Gm. of the dry medium in 1,000 ml. of cold distilled water and heating to boiling to dissolve the medium completely. The addition of 5 Gm. of agar makes the medium more suitable for routine use. The solution is distributed in screw-capped test tubes that are 150 mm. in length, with an outside diameter of 66 mm., and an inside neck diameter of approximately 9 mm.

With the solution in the tubes, and the caps fitted loosely, the tubes are placed in the autoclave for 15 min. at 15 lb. pressure (121 C.). Next the tubed mediums are removed from the autoclave and slanted in such a manner as to allow a generous butt when the medium cools and solidifies.

The heart, liver, spleen, and joint fluids have all proved satisfactory sources for isolating Erysipelothrix from infected turkeys or swine. Using accepted sterile technique, cultures are obtained by stabbing a wire loop into the organ, withdrawing the loop, and inserting it deep into the butt of the triple sugar iron agar medium.

In cases of chronic erysipelas of swine, one of the swollen joints is opened and a sterile cotton swab is used to absorb some of the joint fluid. The swab is then inserted into the butt of the medium and left there. At the time the triple sugar iron agar medium is inoculated, tryptose agar tubes are also streaked along the surface of the slant with a wire loop that has been inserted into one of the internal organs.

All inoculated mediums are then incubated at 37 C. If the inoculum contained Erysipelothrix, hydrogen sulfide begins to form, after six to 36 hours' (av. 14 hr.) in-

From the Clemson College Livestock Sanitary Department diagnostic laboratory, Columbia, S. Car.

*Bacto-Triple Sugar Iron Agar obtained from Difco Laboratories Inc., Detroit, Mich., is used in this laboratory.

Legend for Illustrations on Opposite Page

Fig. 1—(Left to right): Tube 1 shows the medium before inoculation; tubes 2, 3, 4, and 5 show the gradual spread of the hydrogen sulfide to encompass the butt; tube 5 shows the complete color change from the red of the un-inoculated tube to the black butt, the sharp line of demarcation below the water of syneresis, and the yellow slant; tube 6 shows an atypical reaction where the hydrogen sulfide did not spread from the line of the stab, although the red color of the medium has turned yellow.

Fig. 2—(Left to right): Tube 1 shows the medium before inoculation; tube 2 shows the medium 14 hours after inoculation, with the hydrogen sulfide forming along the line of the stab; tube 3 shows the completed reaction 48 hours later (notice the black butt, sharp line of demarcation below the water of syneresis, and the yellow slant); tube 4 shows a completed reaction of Salmonella pullorum; tube 5 shows a completed reaction of paratyphoid spp.; tube 6 shows a completed reaction of Pseudomonas spp. (notice red slant of tubes 4 and 5).

cubation, along the line of the stab where the wire loop was inserted into the butt.

This formation is first noticed as a minute black speck which spreads along the line of the stab within a few hours. Characteristically, this black discoloration cuts off abruptly just before reaching the water of syneresis. At this point, the black discoloration is confined to a narrow zone along the line of the stab. Since phenol red is used in the medium as an indicator to determine the acidity, the medium is red and the black stab stands out in sharp contrast.

Over a period of several hours, the hydrogen sulfide gradually spreads out from the original stab line and eventually encompasses the entire butt, but never extends upward further than the original sharp cut-off just below the water of syneresis. After 24 hours or longer, the red color of the medium that hasn't been discolored black by the hydrogen sulfide gradually turns yellow. The yellow discoloration indicates fermentation of the sugars. No gas is formed. The reactions seem to occur as readily at room temperature as when the medium is incubated. The various color changes produced are shown (fig. 1).

For additional confirmation, the tryptose agar slants are checked for the appearance of minute colonies typical of Erysipelothrix. These are usually found if the typical reaction has occurred on the triple sugar iron agar medium. Occasionally, Erysipelothrix fails to grow on tryptose agar from the original inoculation, even though the triple sugar iron agar has detected its presence. Transfers can readily be made from the triple sugar iron agar to the tryptose agar for confirmation. Further confirmation, if desired, can be obtained by inoculating sugars from the growth on the tryptose agar slants.

Other organisms, including Proteus spp., Salmonella spp., and Pseudomonas spp., form hydrogen sulfide. None of these have been found to give the characteristic reaction of first forming hydrogen sulfide just along the line of the stab and stopping abruptly just below the water of syneresis (fig. 2).

SUMMARY AND CONCLUSION

A method is described for the rapid isolation and identification of Erysipelothrix by the use of triple sugar iron agar. Identification can sometimes be made in six hours, but usually an average of 14 hours is required.

During 12 months, a total of 40 isolations from separate flocks of turkeys and herds of swine was made by the method described. The isolations were confirmed by typical reactions on sugars and other mediums, and by animal inoculation.

The advantage of using triple sugar iron agar in the diagnosis of erysipelas is that it is rapid, economical, reliable, and adaptable to a practitioner's laboratory.

References

[1]Difco Laboratories, Inc.: Difco Manual of Dehydrated Culture Media and Reagents for Microbiological and Clinical Laboratory Procedures. 9th ed. Difco Laboratories, Inc., Detroit 1, Mich. (1953): 166.

[2]Hagan, W. A., and Bruner, D. W.: The Infectious Diseases of Domestic Animals. 2nd ed. Comstock Publishing Co., Inc., Ithaca, N. Y. (1951): 160.

Effect of Season on Erysipelas Infection.—*Erysipelothrix rhusiopathiae* infection was attempted by skin scarification in 180 pigs over a period of three years in Hungary. Rarely was infection established during November to January when the natural incidence is rare; during the rest of the year, the infection rate was 70 to 100 per cent.—*Vet. Bull. (July, 1958): Item 2019.*

Report on Zoonoses

The following items are from the reports *Morbidity and Mortality* issued weekly by the U.S. Department of Health, Education, and Welfare.

Rabies in Bats.—In Florida, at night, 2 bats flew through the window of a car and 1 attacked the driver, inflicting several deep puncture wounds on his hand.—*June 20, 1958.*

In Pennsylvania, a bat entered a window and attacked a man in bed, biting him on the forearm.—*July 5, 1958.*

In both of the above cases, Negri bodies were found in the bats.

In New Mexico, a moribund bat was found to have rabies. About 15 other bats had been found dead or dying in the same vicinity.—*Sept. 5, 1958.*

• • •

Anthrax.—Over 700 cases of anthrax in cattle were reported in ten parishes in northeastern Louisiana, 500 in one parish. Some swine and horses were affected. A

rendering plant employee was also infected.

In southeastern Arkansas, anthrax appeared in 9 herds of cattle in one county and 5 herds in another. In both states, the enzootic areas were under quaratine, vaccination with a noncapsulated anthrax spore vaccine was being carried out, and the disease was subsiding.—*July 5, 1958.*

In Arkansas, 2 cases of anthrax developed in stock handlers, both typical cutaneous anthrax lesions on the hands. Both made complete recoveries when treated.—*July 18, 1958.*

In Arkansas, 2 more cases of anthrax in man were reported. A 7-year-old girl had a typical cutaneous lesion on the thigh, probably the result of an insect bite. A 3-year-old girl had a lesion on the breast where she had fallen on a piece of wood in a lot where 2 horses had died of anthrax.— *Aug. 29, 1958.*

• • •

Arthropod-Borne Encephalitis.—In California, 4 cases of encephalitis in man were reported. Complement-fixation tests indicated that 3 were western equine and 1 was St. Louis encephalitis. During June, when about 50 cases of encephalomyelitis in horses were reported, western equine encephalitis virus was isolated from 38 pools of mosquitoes in California.—*July 25, 1958.*

To September 6, in California, 28 cases of western equine, and 3 of St. Louis encephalitis in man had been reported. The virus of western equine encephalitis was isolated from the brain of a squirrel.—*Sept. 12, 1958.*

In New Mexico, 15 persons, from 2 to 60 years of age, and 7 horses had encephalitis, and one man died. In Utah, there were 20 cases of encephalitis in man, mostly in infants, and four deaths were reported. Several cases in horses were being studied.—*Sept. 12, 1958.*

• • •

Tularemia.—In New Mexico, a 39-year-old rancher, who had hunted and skinned rabbits for years, had a history of fatigue for about a year, a slow-healing (1.5 mo.) laceration on a finger, and a swollen lymph node in the axilla. He had a positive tuleremia agglutination titer of 1:2,560.—*Oct. 10, 1958.*

• • •

Trichinosis.—In Ohio, one hospital reported 78 cases of trichinosis in man.—*Aug. 22, 1958.*

Infectious Diseases in Canada

During the year ending March 31, 1957, no cases of anthrax were reported in Canada. No cases of sheep scab had been reported since 1927. Scrapie was identified on two premises during the year, 1 in Quebec and 1 in Alberta.

Of 179 cases of rabies, mostly in northern Ontario, 9 were in dogs, and 105 in foxes. No human fatalities were reported although many persons had been treated. Approximately 1,500 dogs were vaccinated.

Hog cholera had not been diagnosed in Canada since the year ending in March, 1954. The largest number of cases reported since 1910 was 35,992 in 1941. Since 1946, cholera was reported only in 1952 (112 swine), and in 1954 (3,075 swine).—*Rep. Veterinary Director General, Canada Dept. of Agric., March 31, 1957.*

The number of modified-certified brucellosis-free states rose to 15 when Michigan and New Mexico were added to the list. New Jersey and Utah were the last states added previously.—*Agric. Res. (Sept., 1958): 16.*

Persistence of Agglutination Titer in Calves Vaccinated with Brucella Strain 19.—Tests of over 5,000 blood specimens from heifers in Germany which were vaccinated at 5 to 9 months of age with strain 19 showed 19.5 per cent to be positive and 35.9 per cent doubtful at 18 months of age; 4.3 per cent positive and 19.5 per cent doubtful at 24 months of age; and none positive but 7.1 doubtful at 31 months of age.—*H. Gramatzki and H. W. Teute in Berlin u. Münch. Tierärztl. Wchnschr. (Sept. 1, 1958): 323.*

Radioactive Fallout Concentrated in Fetal Thyroid.—Since iodine-131 from radioactive fallout is concentrated in the thyroid glands of grazing animals, this agent was injected into 2 cows in the ninth month of pregnancy. When killed 24 hours later, the concentration of iodine-131 was six to seven times greater in the thyroid glands of the fetuses than in those of the dams. In other experiments, this concentration apparently began at 53 days of gestation and increased rapidly after the fifth month.—*Science (Sept. 12, 1958): 597.*

Toxoplasmosis in Animals

DANIEL WEINER, M.S., V.M.D.

Atlanta, Georgia

TOXOPLASMA GONDII has been known since 1908, when it was first discovered in a North African rodent, *Ctenodactylus gondii*.[36] Since that time, the organism has been reported from various domestic and wild species all over the world. While the early interest in this organism was purely zoological, indisputable evidence of the occurrence of toxoplasmosis in man was presented in 1939.[50]

Originally, many specific names were given to Toxoplasma from the various hosts; however, all biological and immunological studies have indicated that these parasites are members of the same species, *T. gondii*.

Toxoplasma is typically an intracellular parasite which may attack almost any organ, with a predilection for elements of the reticuloendothelial and central nervous systems. It has also been found free in various tissues.

Its life cycle within the host is relatively simple. After invading the host cell the parasite reproduces, eventually giving rise to hundreds of daughter organisms. The reproduction process was long thought to be binary fission; however, Toxoplasma organisms were recently shown to reproduce by a different and unusual process.[16] Each parent cell develops within itself two daughter cells, small replicates of the parent, which eventually fill the anterior portion of the cell and then break out, destroying the original cell.

In the course of multiplication, this aggregation of parasites is invested in a membrane and is known as a pseudocyst, which may represent the infectious stage that is transmitted to new hosts. Spread of infection within the host's body is effected by the unencysted free parasites, possibly through the blood, in which they can be detected during certain periods of the infection.

The type of tissues affected, the species and age of the host, as well as the strain of parasite, all determine the variable character of the clinical signs and course of toxoplasmosis. The most important clinical signs are encephalitis, febrile exanthema with pneumonitis and enterocolitis.

The organism is widely distributed in nature in many orders of birds and mammals from the most primitive forms to the most highly developed. It has been reported in a marsupial (the wombat);[8] in Insectivora (the mole,[43] shrew, and hedgehog);[31] many rodents (Gondi, mouse, rat, rabbit, guinea pig, squirrel, chinchilla, and marmot); in carnivores (the dog, fox, and cat); artiodactyla such as swine, sheep, and cattle;[4,15,17,27,35,39] and in primates (the baboon,[32] chimpanzee,[26] and several species of monkeys—white-face,[10] marmoset,[11] and rhesus[9]).

Pigeons[21] and chickens[13] have been found naturally infected, while the canary and duck,[51] English sparrow, song sparrow,[33] and the purple grackle[34] have been experimentally infected.

CLINICAL AND PATHOLOGICAL COURSE

The clinical course of toxoplasmosis has been extensively studied in man, in whom the type of disease varies according to whether the infection was contracted *in utero* (congenital), or after birth (acquired or postnatal).

Congenital infections of infants are characterized chiefly by disorders of the central nervous system, with signs and symptoms of necrotizing encephalomyelitis, chorioretinitis, cerebral calcifications, and neurological disturbances.

Acquired infection is manifested in younger children mainly by encephalitis, in adults as an acute febrile exanthema with pneumonitis, but without cerebral involvement. Adults are usually symptomless carriers, in whom the latent infection is revealed only by positive serological reactions or, in the case of mothers, by congenital infection in their offspring.

The pathological changes take the form of multiple proliferative granulomas with signs of necrosis. These are thought to be

From the Department of Health, Education, and Welfare, Communicable Disease Center, Public Health Service, Atlanta, Ga.; Dr. Weiner's present address—Washington State Health Dept., Smith Tower, Seattle, Wash.

due to the elaboration of a powerful exotoxin by the parasites.[45]

In general, the clinical course and pathological changes in animals follow the pattern in humans. A brief description of the disease in domestic animals follows.

TOXOPLASMOSIS IN DOMESTIC ANIMALS

Swine.—In swine, toxoplasmosis is manifested by weakness, coughing, incoordination, and enteritis with diarrhea. Fever is usually absent, except in acute cases. The pathological changes include pneumonia, lymphadenitis, hydrothorax, ascites, ulcerative and fibrinonecrotic enteritis and hepatitis. Necrotic foci have also been found in the brain.[16]

Microscopically, the lesions are characterized by focal necrosis and granulomatous inflammation involving the lungs, lymph nodes, liver, adrenal glands, and spleen. Subependymal, focal, and perivascular microglial granulomas and necrosis are seen in the brain.[7]

Toxoplasma gondii was recovered from the placentas and milk of gilts that had been experimentally inoculated subcutaneously, intramuscularly, and orally, and from 2 newborn pigs farrowed by a naturally infected sow, with no signs of illness.[40] Organisms have also been isolated from edible portions of the carcass.

Cattle.—In cattle, the infection may run an acute course, with fever, dyspnea, coughing, sneezing, nasal discharge, and extreme weakness, as well as central nervous disorders. It is fatal in some cases.

Pathological changes include necrotic foci and calcification in the brain. The parasites have been found in the cerebral lesions, lungs, bronchi, and the spleen. Microscopically, chronic pneumonia, focal and perivascular myocarditis, and necrotic foci of the liver have been found.

Toxoplasma organisms have been demonstrated in the milk as well as in edible portions of the carcass.[39]

Sheep.—Ovine toxoplasmosis is characterized by fever, dyspnea, generalized tremors, incoordination, abortion, and stillbirth. Microscopic necrotizing and granulomatous lesions containing Toxoplasma are widely distributed but are most frequent and severe in the respiratory and central nervous systems. Granulomatous interstitial pneumonia is seen, as well as necrosis, microglial granulomas, vasculitis, and calcifications in the brain and spinal cord.

In utero infection in lambs from ewes that had been experimentally infected was widely distributed and well advanced. Focal necrosis and microglial granulomas with some areas of calcification were demonstrated histologically from the spinal cord and cerebrum.[7]

Dogs.—Young dogs develop an acute disease of short duration while, in adults dogs, the infection is subclinical.[24] Two types of the disease in dogs were described:[17] (*a*) the visceral form, characterized by fever, loss of condition, gastroenteritis with diarrhea and pneumonia, with death occurring in two to ten weeks; and (*b*) the nervous form, manifested by psychic disturbances and locomotor disorders, in some cases resembling distemper and "hard-pad" disease.

In the brain, lesions of meningoencephalitis are found, but no calcification. The parasites invade the intestinal wall, producing ulceration of the mucosa, from which they escape into the lumen and may pass out with the feces, in which the parasites have been detected.[6,24] Congenital toxoplasmosis also occurs in dogs.[7,24]

Cats.—There is little in the literature that gives information on the clinicopathological picture of toxoplasmosis in cats.[19,27] Essentially the disease is similar to that in dogs; however, there is more of a pneumonitis with granulomatous lesions and calcification in the lungs.[27]

Poultry.—In an epizootic among chickens in southeastern Norway the disease was often fatal, and it occasionally caused blindness in birds that survived.[13] The most constant lesions were pericarditis, myocarditis, encephalitis, and ulcerating gastroenteritis. Necrosis of the optic chiasma was also noticed.

Other epizootics have been reported in Norway as well as investigations of endemic disease in flocks from other parts of the world.[2,40] The incubation period of toxoplasmosis in 1-day-old chickens that had been inoculated intraperitoneally or subcutaneously was six to 16 days. The mortality rate was approximately 50 per cent.[38]

EPIDEMIOLOGY

The only method of transmission of toxoplasmosis in man and lower animals that has been established beyond all doubt is the congenital one. In congenital cases in man, the transmission is made from a chronic tolerant carrier to the infant.[45]

Presumably, pregnancy may result in an exacerbation of latent toxoplasmosis and the spread of parasites from a localized site of chronic infection to the placenta and fetus. Probably the parasites first invade the placenta and then infect the fetus, since the organisms have been found in the uterine wall and in the placenta.[7,39,40]

In its proliferative form, *T. gondii* manifests little resistance to environmental factors outside the body of the host. It is rapidly destroyed by drying, changes in osmotic pressure, low heat, and freezing and thawing, except under special conditions.[5,14] A rapid attrition of organisms occurs when they are permitted to stand outside of cells in saline or serum-saline solutions, or in tissue culture nutrients.[22]

There is a similar attrition of the proliferative forms in the carcasses of infected animals. Although such material stored in the refrigerator for two weeks may still be infective for mice, the survival time of these animals is definitely prolonged.[25] Because of the lack of resistance of the proliferative forms of the parasite, in all probability infection is not established by contaminative means.

Droplet transmission from infected animals with pulmonary involvement and rhinitis is possible, since free pseudocysts have been found in the lumina of the alveoli and bronchioles, from which they can be discharged with the saliva.[30] Nasal secretion of infected rabbits was infective to other animals.[26] In herbivorous animals, the infection could be acquired from grass contaminated with saliva or nasal discharge of infected animals. Rodents became infected after exposure to sprays of vaporized suspensions of Toxoplasma organisms.[29]

Regarding insect transmission, experiments have indicated that blood-sucking ectoparasites and other arthropods can not be vectors of toxoplasmosis.[48] Of 17 species of blood-sucking arthropods studied, the only positive results were related to three ticks, *Dermacentor variabilis, Dermacentor andersoni,* and *Amblyomma americanum,* and to the body louse *Pediculus humanus corporis* in man.[49] Transmission occurred only in exceptional instances, and positive results were not obtained consistently.

Attempts to incriminate arthropods in the transmission of the infection have been unsuccessful. Inasmuch as epidemiological data indicate the presence of the parasite in urban and rural areas, among groups of considerably different socioeconomic status, and in all areas throughout the world, it is difficult to conceive of a single arthropod species as the sole vector.

Experiments on the oral route of infection have brought conflicting results. One investigator was able to pass the infection consistently by the oral route when he used the tissues of chronically infected animals.[12] There has been little success in transmitting infection by feeding material from mice that were dead or dying from toxoplasmosis.[20] More consistent results were obtained when material from chronically infected animals was fed. This was probably due to the presence of the more resistant pseudocyst forms in the chronically infected animals.

Recently, it was suggested that transmission through ingestion of pork may occur inasmuch as mice, rats, pigs, and primates were infected by the oral route.[46] However, positive dye test titers have been found among orthodox Jews, who denied ever having eaten pork, as well as in vegetarians, who had never consumed meat of any kind.[23] The role of beef in the transmission of toxoplasmosis has not been studied.

Since there is no host restriction, Toxoplasma infection is interchangeable between a wide range of animals, including man. Theoretically, it is conceivable that any infected animal might be a source of human infection. It is obvious that the risk of infection from animals is greatest from those in the environment of man, and especially those with which man comes in close contact. Although no single animal species has been incriminated as yet, there is considerable circumstantial evidence that rabbits, dogs, farm animals, birds, and perhaps some game animals might act as reservoirs of the disease.

It was showed that in Sheffield, England, the proportion of cases in man was significantly higher among persons handling rabbits or their carcasses (e.g. veterinarians, abattoir workers, trappers) than in the general population.[1] A definite correlation has been demonstrated between infections in man and dogs in the same household,[6,47] but whether man acquires the infection directly from dogs or both become infected from a common source is not clear.

It may be significant that in outbreaks

among hares and capercaillies (a large European grouse), in Sweden, the geographical distribution of toxoplasmosis in man and game coincided.[3]

DIAGNOSIS AND CHEMOTHERAPY

The clinical characteristics of toxoplasmosis are so variable that clinical diagnosis is difficult. Laboratory procedures should, therefore, be relied upon for assistance in diagnosis.

The following are postulated conditions in which laboratory procedures are indicated:[42] (1) fever of unknown origin; (2) lymphadenopathy; (3) encephalitis; (4) exanthematic diseases; (5) myocarditis; and (6) chorioretinitis.

Serological tests are indicated in all suspected cases of toxoplasmosis and initial dependence should be placed on the methylene blue dye test because of the relatively late appearance of a titer in the complement-fixation test. Actual confirmation of the diagnosis may be obtained only through isolation of the parasite from the tissues of the patient via mouse inoculation.

The intradermal test is of little value as a diagnostic procedure in man because of the relatively long period required for the development of fixed antibodies.[42] In animals, the available antigen is too weak to elicit a postive reaction from infected individuals.[44]

Extensive screening tests with a large number of drugs in experimental animals have indicated that two groups of drugs have some activity against *T. gondii*. These are: (1) the sulfonamides, the most active of which are the sulfapyrimidines (sulfamethazine, sulfamerazine, and sulfadiazine) and sulfapyrazine; and (2) 2, 4-diamino-pyrimidines, the most active of which is pyrimethamine.[15]

Best results are obtained from a combination of members of the two groups, as it has been demonstrated that they act synergistically. While the exact nature of the therapeutic response is not known, it is probable that the combination of drugs tends to limit proliferation of the parasites within the host. There is evidence that the drugs act only against proliferating parasites and not against those in pseudocysts.

CONCLUSION

Although many workers have studied the various aspects of toxoplasmosis, there still remain many gaps in our knowledge. The mechanics of transmission, easier methods of diagnosis, and a better means of treatment are needed to round out our knowledge of this ubiquitous disease.

References

[1]Beverly, J. K. A., Beattie, C. P., and Roseman, C.: Human Toxoplasma Infection. J. Hyg., *52*, (1954): 37-46.

[2]Biering-Sorenson, U.: Fjerkraetoxoplasmose. On forekomsten af endemisk optraedende toxoplasmose i danske honsebesaetninger. Nord. Vet.-med., *8*, (1956): 140-164.

[3]Borg, K.: Toxoplasmosis in Hares and Capercaillie in Sweden During the Years 1948-1952. Proc. Fifteenth Internat. Vet. Cong., Stockholm, *1*, (1953): 406-409.

[4]Callahan, W. P., Jr., Russell, W. O., and Smith, M. G.: Human Toxoplasmosis. A Clinico-Pathologic Study with Presentation of 5 Cases and Review of the Literature. Medicine, *25*, (1946): 343-397.

[5]Chandler, A. H., and Weinman: Prolonged Storage of Toxoplasma at −70 C. A Preliminary Report. Am. J. Clin. Path., *26*, (1956): 323-326.

[6]Cole, C. R., Prior, J. A., Docton, F. L., Chamberlain, D. M., and Saslaw, S.: Toxoplasmosis. III. Study of Families Exposed to Their Toxoplasma Infected Pet Dogs. Am.M.A. Arch. Int. Med., *92*, (1953): 308-313.

[7]Cole, C. R., Sanger, V. L., Farrell, R. L., and Kornder, J. D.: The Present Status of Toxoplasmosis in Veterinary Medicine. North Am. Vet., *35*, (1954): 265-270.

[8]Coutelan, F.: Existence d'une encéphalite toxoplasmique spontanée chez les Wombats. Un toxoplasme nouveau *Toxoplasma wenyoni* n. sp., parasite de *Phascolomys mitchelli* (Australia). Compt. rend. Soc. Biol., *110*, (1932): 1245-1247.

[9]Cowen, D., and Wolf, A.: Toxoplasmosis in the Monkey. Acute Fatal Infection Experimentally Produced in a Young *Macaca Mulatta*. J. Infect. Dis., *77*, (1945): 144-157.

[10]DeRodaniche, E.: Spontaneous Toxoplasmosis in the Whiteface Monkey, *Cebus Capucinus* in Panama. Am. J. Trop. Med. Hyg., *3*, (1954a): 1023-1025.

[11]DeRodaniche, E.: Susceptibility of the Marmoset *Marikina Geoffroyi* and the Night Monkey *Aotus Zonalis* to Experimental Infection with Toxoplasma. Am. J. Trop. Med. Hyg., *3*, (1954b): 1026-1032.

[12]Eichenwald, H.: Experimental Toxoplasmosis. I. Transmission of the Infection in Utero and Through the Milk of Lactating Female Mice. Am. J. Dis. Child., *76*, (1948): 307-315.

[13]Erickson, S., and Harboe, A.: Toxoplasmosis in Chickens. I. An Epidemic Outbreak of Toxoplasmosis in a Chicken Flock in Southeastern Norway. Acta. path. et. microbiol. Scand., *33*, (1953): 56-71.

[14]Eyles, D. E., Coleman, N., and Cavanaugh, D. J.: Preservation of *Toxoplasma Gondii* by Freezing. J. Parasitol., *42*, (1956): 408-413.

[15]Eyles, D. E.: Newer Knowledge of the Chemotherapy of Toxoplasmosis. Ann. New York Acad. Sci., *64*, (1956): 252-267.

[16]Farrell, R. L., Docton, F. L., Chamberlain, D. M., and Cole, C. R.: Toxoplasmosis. I. Toxoplasma

Isolated from Swine. Am. J. Vet. Res., *13,* (1952): 181-185.

[17]Frankhauser, R.: Toxoplasmose beim Hund. Schweiz. med. Wchnschr., *81,* (1951): 336-338.

[18]Goldman, M., Carver, R. K., and Sulzer, A. J.: Similar Internal Morphology of *Toxoplasma Gondii* and *Besnoitia Jellisoni* Stained with Silver Protein. J. Parasitol., *43,* (1957): 490-491.

[19]Holzworth, J.: Toxoplasma Infection in Cats. J.A.V.M.A., *124,* (April, 1954): 313.

[20]Jacobs, L., Woke, P. A., and Jones, F. E.: Studies on the Transmission of *Toxoplasma Gondii.* J. Parasitol., *36,* (1950): 36-37.

[21]Jacobs, L., Melton, M. L., and Jones, F. E.: The Prevalence of Toxoplasmosis in Wild Pigeons. J. Parasitol., *38,* (1952): 457-461.

[22]Jacobs, L., Jones, F. E., and Melton, M. L.: The Survival of *Toxoplasma Gondii* in Various Suspending Media. J. Parasitol., *38,* (1952): 293-297.

[23]Jacobs, L., Cook, M. K., and Neumann, E.: Serological Survey Data on the Prevalence of Toxoplasmosis in the Jewish Population of New York. J. Parasitol., *40,* (1954): 701-702.

[24]Jacobs, L., Melton, M. L., and Cook, M. K.: Observations on Toxoplasmosis in Dogs. Parasitology, *41,* (1955): 353-361.

[25]Kass, E.: The Viability of Toxoplasma in Mouse Carcass. Acta. path. et microbiol. Scand., *37,* (1955): 84-88.

[26]Kass, E., and Steen, E.: Serological Investigations of Rabbits Experimentally Infected with *Toxoplasma Gondii.* Acta. path. et microbiol. Scand., *28,* (1951): 169-173.

[27]Keagy, H. F.: Toxoplasma in the Chinchilla. J.A.V.M.A., *114,* (Jan., 1949): 15.

[28]Kopciowska, L., and Nicolau, S.: Toxoplasmose spontanée du chimpanzé. Compt. rend. Soc. Biol., *129,* (1938): 179-181.

[29]Kunert, H., and Schmidtke, L.: Inhalationsversuche mit *Toxoplasma Gondii.* Ztschr. Tropenmed. u. Parasitol., *5,* (1954): 324-329.

[30]Lainson, R.: Studies on the Epidemiology of *Toxoplasma Gondii* in England. II. Variation Factors in the Pathogenesis of Toxoplasma Infections. Ann. Trop. Med. Parasitol., *49,* (1955): 397.

[31]Laveran, A., and Marullaz, M.: Recherches expérimentales sur le *Toxoplasma Gondii.* Bull. Soc. path. Exot., *6,* (1913): 460-468.

[32]Levaditi, C., and Schoen, R.: Présence d'une toxoplasmose dans l'encéphale du *Cynocephalus Babuin.* Bull. Soc. path. Exot., *26,* (1933): 402-405.

[33]Manwell, R. D., Coulston, F., Binckley, E. C., and Jones, V. P.: Mammalian and Avian Toxoplasmosis. J. Infect. Dis., *76,* (1945): 1-14.

[34]Manwell, R. D., and Drobeck, H. P.: Mammalian Toxoplasmosis in Birds. Exptl. Parasitol., *1,* (1951): 83-93.

[35]Moller, T.: Toxoplasmosis Vulpis Vulpis. Acta. path. et microbiol. Scand. Suppl., *93,* (1952): 308-320.

[36]Nicolle, C., and Manceaux, L.: Sur une infection à corps de Leishman (ou organismes voisins) du gondi. Compt. rend. Acad. Sci., *147,* (1908): 763-766.

[37]Olafson, P., and Monlux, W. S.: Toxoplasma Infection in Animals. Cornell Vet., *32,* (1942): 176-190.

[38]Qureshi, M. A. A.: Experimental Toxoplasmosis in the Chicken. Proc. Sixth Pakistan Sci. Cong., Part III. Karachi (1954): 226.

[39]Sanger, V. L., Chamberlain, D. M., Chamberlain, K. W., Cole, C. R., and Farrell, R. L.: Toxoplasmosis. V. Isolation of Toxoplasma from Cattle. J.A.V.M.A., *123,* (Aug., 1953): 87-91.

[40]Sanger, V. L., and Cole, C. R.: Toxoplasmosis. VI. Isolation of Toxoplasma from Milk, Placentas, and Newborn Pigs of Asymptomatic Carrier Sows. Am. J. Vet. Res., *16,* (1955): 536-539.

[41]Schulte, F.: Toxoplasmose. Encephalitis beim Geflügel. Deutsche tierärztl. Wchnschr., *61,* (1954): 481-484.

[42]Siim, J. C.: Toxoplasmosis Acquisita Lymphonodosa: Clinical and Pathological Aspects. Ann. New York Acad. Sci., *64,* (1956): 185-206.

[43]Von Prowazek, S.: Parasitische Protozoen aus Japan. Gesammelt von Hern Dr. Mine in Fokuoka. Arch. f. Schiffs—u. Trop. Hyg., *14,* (1910): 297-302.

[44]Weiner, D.: Unpublished data.

[45]Weinman, D.: Toxoplasma and Toxoplasmosis. Am. Rev. Microbiol., *6,* (1952): 281-298.

[46]Weinman, D., and Chandler, A. H.: Toxoplasmosis in Man and Swine. An Investigation of the Possible Relationship. J.Am.M.A., *161,* (1956): 229-232.

[47]Westphal, A., and Finke, L.: Der Hund als epidemiologischer Faktor der Toxoplasmose des Menschen. Ztschr. Tropenmed. u. Parasitol., *2,* (1950): 236-239.

[48]Weyer, F.: Ein experimenteller Beitrag zur Frage der Ubertragung von *Toxoplasma Gondii* durch Arthopoden. Ztschr. Tropenmed. u. Parasitol., *3,* (1951): 65-72.

[49]Woke, P. A., Jacobs, L., Jones, F .E., and Melton, M. L.: Experimental Results on Possible Arthropod Transmission of Toxoplasmosis. J. Parasitol., *39,* (1953): 523-532.

[50]Wolf, A., Cowen, D., and Paige, B. H.: Toxoplasmic Encephalomyelitis. III. A New Case of Granulomatous Encephalomyelitis Due to a Protozoon. Am. J. Path., *15,* (1939): 657-694.

[51]Wolfson, F.: Mammalian Toxoplasma in Erythrocytes of Canaries, Ducks and Duck Embryos. Am. J. Trop. Med., *21,* (1941): 653-658.

[52]Wright, W. H.: A Summary of the Newer Knowledge of Toxoplasmosis. Am. J. Clin. Pathol., *28,* (1957): 1-17.

Transmission of Toxoplasmosis

In experiments in Brazil, mice inoculated with contents of the digestive tube of the arthropod triatomid, fed up to three days earlier with infected material, usually died within ten days with acute toxoplasmosis. The maximum period of survival of Toxoplasma in this arthropod was 12 days.

Toxoplasmosis was not transmitted when infected triatomids were fed to the mice or when the mice were inoculated with eggs from infected triatomids.—*J. Am. M. A. (Aug. 9, 1958): 1865.*

Experimental Bovine Leptospirosis.—Experimental infection of yearling bulls with *Leptospira sejroe* caused fever, slug-

gishness, diarrhea, and stiffness of the joints. Bacteremia persisted for up to six days after the onset of signs of infection. The micro-tube agglutinin titer was 1:64 six days after infection, with a maximum of 1:16,000 at ten to 12 days.—*Vet. Bull. (Sept., 1958): Item 2766.*

Fetal Leptospirosis and Abortion

Fetal leptospirosis has been accepted as the cause of abortion in swine but is questioned in cattle. In an experiment in Norway, after laparotomy, living cultures of either *Leptospira pomona* or *Leptospira sejroe* were each injected into the placetomes of 2 pregnant, immunized cows. Three aborted 12 to 14 days later and leptospiras were demonstrated in silver-impregnated sections of liver and kidney from all 3, although the organism was not isolated. The fourth animal in which the fetus died on the sixth day, was killed a few hours later and the fetus was recovered. The fetus had died from leptospirosis and *L. pomona* was isolated from all organs and body fluids examined.

It is concluded that abortions follow fetal leptospirosis due to leptospiral penetration of the placenta during maternal leptospiremia. The two consecutive infections would explain the interval of two to five weeks between exposure of the dam and abortion. Failure to isolate the organism from aborted material could be because the interval between fetal death and abortion exceeds the survival time of the leptospiras in the dead fetus.—*K. L. Fennestad and C. Borg-Petersen in J. Infect. Dis. (May-June, 1958): 227-236.*

Leptospira Antibody in Bovine Fetuses.—It is commonly believed that the bovine fetus is unable to produce antibody. After laparotomy, 5 ml. of *Leptospira saxkoebing* suspension was injected directly into the placetomes of 2 cows, pregnant 214 and 223 days. To prevent maternal leptospirosis, the cows had been injected intravenously, 24 hours previously, with anti-leptospirosis serum. The calves were born 47 and 41 (twins) days later, respectively, and the membranes were expelled within eight hours. When killed nine and seven days later, the 3 calves showed no gross lesions but did show histological evidence of leptospirosis, although Leptospira could not be demonstrated. The titers of the pre-colostral serums of the calves greatly exceeded the titers of the dams and continued to rise, indicating prenatal infections.—*K. L. Fennestad and C. Borg-Petersen in Nature (Nov. 30, 1957): 1210.*

Bovine Fetal Leptospirosis in the United States.—Gross and histopathological evidence of bovine fetal leptospirosis in 6 fetal specimens is presented as evidence that leptospiral infection of the fetus is a major cause of the abortions in bovine leptospirosis. The author reports no difficulty in demonstrating leptospiras in fetal tissues by silver-impregnation methods.—*C. H. Bridges in Southwest. Vet. (Summer, 1958): 271.*

Anthrax and Bone Meal.—Of 9 cases of anthrax in human beings in Scotland, 4 were in factory workers handling charcoal used in sugar refining and made from bones imported from India, Pakistan, and Argentina. Spores of virulent anthrax bacilli were found in the bone dust, even in the factory offices. Four cases were in farm laborers handling bone meal fertilizer. The ninth was in a veterinarian who had contracted the disease from an infected animal.—*J. Am. M. A. (Sept. 20, 1958): 314.*

Aftosa in A Grizzly Bear.—Foot-and-mouth disease, or aftosa (a disease of "cloven-hoofed" animals), was diagnosed, on July 17, 1948, in a grizzly bear in the Buenos Aires zoo. He seemed weak, had no appetite, and moved with difficulty. A young bear in the same cage was similarly affected the next day. Both developed vesicles on the tongue and also large ones, which soon ruptured, on the hindfeet. The animals recovered in about ten days.—*A. M. Grosso in Gaceta Veterinaria (March, 1958): 72.*

Survival of Aftosa Virus in Bacon.—Bacon from swine slaughtered at the height of infection remained infective after 55 days of storage at −15 to −20 C. Thawing did not reduce the infectivity, but the virus died about five days after thawing, when the pH had fallen below 6.—*Vet. Bull. (Sept., 1958): Item 2820.*

New Two-Compartment Disposable Plastic Syringe

JULES SILVER, V.M.D.

North Franklin, Connecticut

A unique disposable plastic syringe, filled and ready for use with the dry powder and diluent stored separately, is now available (fig. 1).

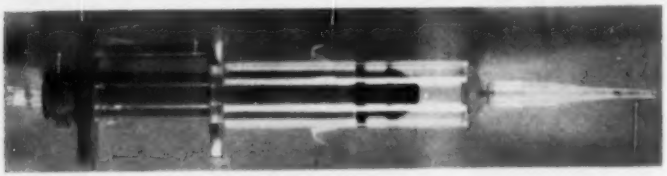


Fig. 1—A disposable plastic syringe, filled and ready for use with the dry powder and diluent stored separately.

This new unit actually serves as a container for the dry product with its diluent and as an instrument for administration (fig. 2).

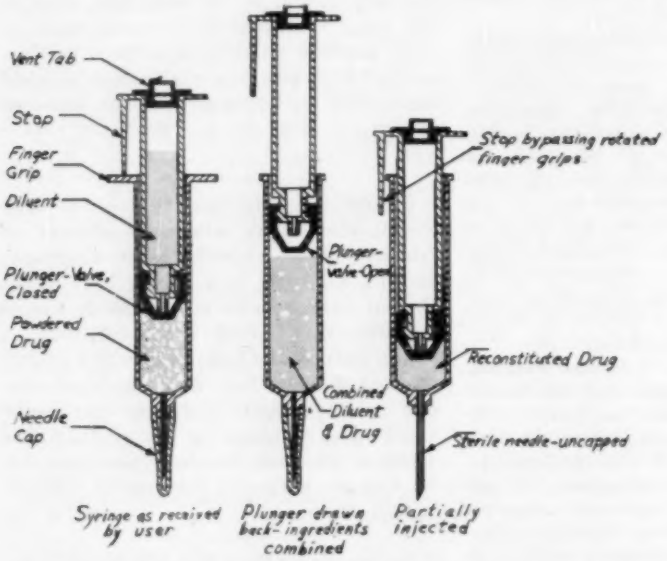


Fig. 2—Component parts of the new disposable plastic syringe (model 1). This unit serves both as a container for the dry product with its diluent and as an instrument for injection.

This disposable syringe is easy to use and assures absolute sterility of the product when injected. It helps to assure the manufacturer that his product will be administered as recommended.

Dr. Silver is a general practitioner in North Franklin, Conn.

More and more pharmaceutical and biological products are being marketed in the dry or lyophilized state with an accompanying diluent in a separate or adjacent vial. The prime reason is that products which must be administered in the fluid state are more stable when stored in dry or desiccated form.

To restore the dry ingredient, the vent tab is removed and discarded. Turning the handle, which serves as a diluent chamber, one quarter turn to the left and drawing it back allows the diluent to rush from the handle through the one-way valve into the barrel containing the dry solid. The reconstituted product is then injected. Intravenous injections can be made as with a conventional syringe.

Summary

The importance of parenteral administration in modern pharmacotherapy calls

for modernized materials and methods for speedy economical use of drugs. A disposable plastic syringe is now available which contains the dry powdered drug in one compartment and the diluent in another, ready for instant mixing and adminstration. Sterility is assured, and the hazards of contamination by transfer from vial to syringe are avoided.

Anesthetic Barbiturate Combinations

A combination of equal parts of hexobarbital and thialbarbitone retains the desirable recovery characteristics of thialbarbitone, regardless of the length of anesthesia, and the anesthesia appears to be at least additive. The duration of anesthesia is intermediate to that of each component.—[*Lawrence C. Weaver, George R. Burch, Benedict E. Abreu, and Alice B. Richards: A Study of the Anesthetic Properties of Combinations of Hexobarbital Sodium and Thialbarbitone Sodium in Dogs. Am. J. Vet. Res., 19, (Oct., 1958): 940-942.*]

Uterine Atony and Hormone Therapy

Two ewes, thought to be lambing but showing no labor, were found to have atony of the uterus and failure of the cervix to dilate (so-called "ring womb"). The cervix would admit a finger, and a piece of ruptured fetal membrane was protruding.

Each ewe was given 150 ml. of 20 per cent borogluconate solution subcutaneously, and 4 ml. (40 units) of posterior pituitary extract and 0.5 ml. (5 mg.) of stilbestrol diproprionate intramuscularly. Both ewes then delivered live lambs normally within 12 hours. This treatment has been successful in several similar cases.—*J. P. Pickering in Vet. Rec. (July 19, 1958): 592.*

Enzootic Abortion in Sheep Due to Salmonella Abortus Ovis—In northern Greece, during 1955 to 1958, *Salmonella abortus ovis* was found responsible for abortions in several herds of sheep. From 25 to 75 per cent of the pregnant females aborted during the last month of gestation, but no abortions occurred during the following year. Because of retained afterbirths, the mortality was up to 11 per cent of the aborting ewes.

Of five common antibiotics tested *in vitro*, the organism was highly sensitive to only streptomycin and chloramphenicol. Therapeutic tests with streptomycin gave encouraging results.—*T. Christodoulou et. al. in Bull. Hellenic Vet. Med. Soc., 30, (1958): 62.*

Seasonal Nature of Parturient Paresis

During a two-week period in June, 1958, more than 20 cases of parturient paresis occurred in cows and heifers within a 12-mile radius, in an area in Scotland. Calcium therapy was not always effective. Poor rations during the winter was believed to be an etiological factor in the heifers. The condition was most pronounced on highly-farmed land.—*Vet. Rec. (July 26, 1958): 610.*

Bacterial Flora of Boar Semen

The uncontaminated ejaculum from a boar contained few bacteria but there were many organisms in the preputial diverticulum. In 9 of 10 boars, the preputial organisms were mostly Pseudomonas but, in 1 animal, there were several types including streptococci, micrococci, Proteus, and corynoform bacteria.—*N. Koppang and O. Filseth in Nord. Vet.-med. (Sept., 1958): 603.*

Treatment of Vibrio Fetus Infection in Bulls.—After epidural anesthesia in standing bulls, treatment was applied locally to the protracted penis and preputial mucosa using, in 30 bulls, trypaflavine solution and ointment and, in 85 bulls, fat-free carbowax ointment in which aqueous solutions of streptomycin and oxytetracycline were incorporated. Control tests (cultures from heifers or from preputial fluid) were made ten to 30 days after treatment and, in several bulls, were continued at intervals up to ten months. This treatment is simple, cheap, and effective in almost all cases when applied carefully.—*Vet. Bull. (Sept., 1958): Item 2779.*

Effects of Thorotrast Myelography.—Thorotrast (25% solution of thorium dioxide) is now infrequently used in man but is still used in canine myelography. In a histological study of the meninges of 12 dogs in which it had been used, the salient pathological effects were phagocytosis and a rapidly fibrosing leptomeningitis.—*Vet. Bull. (Sept., 1958): Item 2985.*

Ruminant Ketosis—Deficiency Disease or Metabolic Defect?

ROBERT B. JOHNSON, A.B.

College Park, Maryland

THE INVESTIGATORS of ketosis have, perhaps, been involved in more than their share of controversy. This has probably occurred because we have long been faced with two apparently contradictory facts: (1) An animal on a diet high in carbohydrate is subject to a disorder characterized by a hypoglycemia; and (2) the disorder is alleviated by glucose administered parenterally. Until these facts were reconciled, no view concerning the cause of ketosis could be entirely rational and, consequently, speculation could proceed in almost any direction.

In recent years, we have witnessed rapid advances in basic knowledge concerning ruminant digestion and intermediary metabolism. The question of ketosis is still controversial, but there is a much broader base of agreement than there was a few years ago. The present controversies are concerned[5,11,13,22,23] more with a degree of emphasis than with diametrically opposed concepts.

AREAS OF AGREEMENT

There now seems to be rather general agreement that the ruminant absorbs little carbohydrate from its digestive tract. The microorganisms of the rumen attack all carbohydrates, using them as a source of energy. The residues of their anaerobic metabolism are acetic acid, propionic acid, butyric acid, and small quantities of higher fatty acids, plus methane and carbon dioxide. It is from these residues of microbial action, and from the microorganisms themselves, that the ruminant obtains the materials required to satisfy its own metabolic needs.

There is general agreement that propionic acid is used for the net synthesis of glucose.

There is also general agreement that, if the ruminant is dependent almost entirely upon propionic acid for its carbohydrate, its supply of carbohydrate may be deficient during periods of high demand.

The apparent anomaly, with which we have been so long plagued, is therefore resolved. Ruminants, although they ingest large amounts of carbohydrates, do not absorb large amounts of carbohydrates from their digestive tract. The lack of response of animals with ketosis to oral carbohydrate, as contrasted with parenteral carbohydrate, is to be expected.

AREAS OF DISAGREEMENT

There is no general agreement on the question of whether acetic acid, butyric acid, or long-chain fatty acids can be used for the synthesis of significant quantities of carbohydrate.

Shaw and his co-workers feel that it is in this area that the ketotic ruminant differs from its normal counterpart. They advance the thesis that in ketosis there is a failure to convert adequate amounts of butyric acid, fat, or protein into carbohydrate.[23,25] They have presented no direct experimental evidence demonstrating a metabolic abnormality in the use of any of these substances.

The problem of whether mammals are able to form carbohydrate from acids containing an even number of carbon atoms has a long and controversial history. In recent years, the prevailing opinion has been that such a conversion does not occur to any significant degree.[26]

California workers have administered various substances, labeled with radioactive carbon, to lactating cows. When acetic or butyric acids are given, the labeled carbon atoms appear not only in the fatty acids of the milk, but also in the lactose, protein, and the glycerol portion of the fat. They state that acetic acid and butyric acid are 'precursors' of these substances and define a precursor as "a substance . . ., a part or all of which is incorporated into a product. . . ."[15].

In other words, if the labeled carbon atom of any substance appears in a second

Mr. Johnson is associate professor of Veterinary Physiology, Department of Veterinary Science, University of Maryland, College Park.

Presented before the General Practice Section, Ninety-Fifth Annual Meeting, American Veterinary Medical Association, Philadelphia, Pa., Aug. 18-21, 1958.

Scientific Article No. A697; contribution No. 2920 of the Maryland Agricultural Experiment Station, Department of Veterinary Science, University of Maryland.

substance, the first substance is considered a precursor of the second.

It is important to recognize that this does not infer that any net synthesis of the second substance from the first occurs. The labeling of lactose can occur without a net synthesis from acetate or butyrate having taken place by the cyclic pathway by which acetic acid and butyric acid are thought to be oxidized. The extent of labeling is an indication of the rate at which acetic acid and butyric acid are being oxidized while lactose is being synthesized from other substances, such as propionic acid. The mechanisms of these reactions are discussed in detail elsewhere.[26]

A recent paper[6] indicates that the labeling observed in the milk constituents, when labeled acetate is administered, is entirely consistent with this pathway by which no net synthesis from acetate results. Although in earlier reports[14,19] these authors indicated that butyrate can be used for the net synthesis of carbohydrate, no study such as the excellent one with acetate has yet appeared.

The evidence reported from work with radioisotopes does not demonstrate that butyrate can be used for the net synthesis of carbohydrate, although it does show that the pathways by which butyrate is metabolized are not completely understood.

In the absence of definitive experiments with radioisotopes, experiments of a more prosaic nature must be considered. A number of recent papers[3,7,12,21] make it clear that when butyric acid is administered orally to starved ruminants no significant rise in blood glucose occurs. Thus, butyric acid can not be a significant precursor* of glucose in tissues that can release glucose to the blood. The possibility that tissues other than liver and kidney can use butyrate for glycogenesis has not been entirely excluded.

There is general agreement that a significant portion of protein can be converted to glucose. There is evidence, however, that the quantity of amino acids that the ruminant can derive from its diet, subsequent to microbial attack, is limited[2,9,16] and that the ruminant's powers of gluconeogenesis from protein are also limited.[11] In any event, the use of protein as a source for glucose is an expensive and wasteful process. We should look elsewhere for dietary precursors of glucose.

*A precursor of a substance, as used in this paper, is defined as a material that can be used for the net synthesis of that substance.

THEORIES CONCERNING CAUSE OF KETOSIS

A Deficiency Disease.—We are thus led to the conclusion that the available evidence is entirely consistent with the interpretation that the ruminant is primarily dependent upon propionic acid for its carbohydrate requirements and that this supply is limited.

There is ample evidence that glucose administered parenterally or glucose precursors, such as glycerol, propionic acid, propylene glycol, and lactic acid, given orally bring about recovery from ketosis. It has recently been demonstrated[20] that relatively small amounts of sodium propionate incorporated into the feed of the newly freshened cow can lead to a significant decrease in the incidence of the disorder.

It is thus clear that evidence of a deficiency state exists and that replacement therapy leads to a remission of the condition.

Two objections have been raised to this nutritional interpretation of ketosis: (1) The blood glucose level in animals with ketosis is not invariably low; and (2) fasting ketosis differs in some respects from clinical ketosis.[23]

In regard to the first point, normal blood glucose levels in cows with ketosis are the rare exception rather than the rule. The blood level of an essential nutrient often reflects a deficiency state in other tissues, but need not invariably do so. For example, there is little correlation between blood nicotinic acid level and the degree of nicotinic acid deficiency.[10]

A recent review[5] has dealt with the second objection, pointing out that starvation is not the same thing as a propionic acid deficiency. Starvation has never been an accepted method of producing the characteristic signs of a deficiency due to the lack of a specific nutrient. Multiple vitamin deficiencies may exhibit an intensification of certain signs and symptoms when all except one of the deficiencies are remedied.[8]

In multiple amino acid deficiencies, "The addition of a small amount of the second most limiting amino acid may increase the severity of the deficiency of the most limiting amino acid."[8] The differences between starvation ketosis and clinical ketosis may be due to a similar relationship.

Let us then consider ketosis a deficiency disease—specifically, lack of adequate carbohydrate precursors in the diet. Is this concept of value in understanding the disorder?

It is well known that ketosis is often a herd problem and that a herd may be affected one year but not the next. Such occurrences lead one to suspect dietary variation. Supporting this suspicion is the frequent association of the use of butyric acid-type silage with a high incidence of ketosis.[1] The seasonal nature of ketosis may also be due to dietary variation.

A Metabolic Defect.—Other manifestations of the disease must be attributed to individual variability. Deficiency diseases are noted for the variability in the signs that they display and in the degree of deficiency required to evoke these signs. This variability is not surprising. The enormous variety which occurs among individuals has recently been re-emphasized.[27] Carbohydrate is an essential nutrient which is utilized in many different ways. It is acted upon by many enzymes. Its rate of metabolism and the pathways that it follows are regulated by several hormones. Individual variation is to be expected at every step of its complex metabolism.

Any situation that leads to an increased demand for an essential nutrient can precipitate deficiency signs. Hence the close correlation, in cows, of the occurrence of ketosis with the stage of lactation, with persistency of lactation, and with the absolute amount of milk produced. Hence the correlation, in sheep, with the gestation of twin lambs.

The correlation with obesity is interesting. When animals are fattening, carbohydrate is being converted to fatty acids. If this conversion continues when carbohydrate is needed for other purposes, it becomes a wasteful process which may lead to deficiency signs. It would be interesting to know if this actually occurs.

It may be true that some animals displaying the signs of ketosis are less able to mobilize their limited protein reserves for conversion to carbohydrate than are normal animals. However, there is no evidence of such a defect.

Of particular concern are the cases, usually encountered with high and persistent producers, that fail to respond well to any of the usual methods of treatment. Almost certainly, these animals are using carbohydrate at an unusually high rate. It would be helpful to know the specific metabolic defect or defects which are present.

Whether we consider ketosis a deficiency disease or a metabolic defect depends upon where we wish to place the emphasis. The evidence certainly seems sufficient to warrant the conclusion that a deficiency state occurs. Metabolic defects probably also occur, but as yet no evidence demonstrating a primary metabolic defect has been presented. The deficiency state would seem to be the underlying factor in all cases of ketosis. It is unlikely that one specific metabolic defect is a contributing factor in more than a portion of the cases which occur.

Adrenal Insufficiency.—It was proposed, in 1950, that bovine ketosis is due to an "adrenal insufficiency."[22] The finding that cows with ketosis exhibit high 17-hydroxycorticosteroid levels in the blood plasma does not support this proposal.[18] The concept has since been modified[23,25] with the use of the term "relative adrenal insufficiency." As was stated previously, no metabolic defect—one controlled by hormones or of any other type—has yet been demonstrated.

OUTLOOK

The final confirmation of the hypothesis presented in this paper depends upon the elimination of the problem of ketosis by manipulation of the diet. The work of Schultz[20] is a preliminary step in this direction. Unfortunately, controlling the products of rumen fermentation has proven to be difficult. Many workers have shown that the proportion of the volatile acids found in the rumen does vary with the diet.[4,17,24]

It is hoped that the realization that ruminant ketosis is fundamentally a deficiency disease will give added impetus to these investigations. It does not seem unduly optimistic to predict that practical diets, producing greater amounts of propionic acid in the rumen, will soon be available.

References

[1]Adler, J. H.: Grass Silage a Possible Nutritional Factor in Bovine Ketosis. Cornell Vet., *46*, (1956): 446-451.

[2]Annison, E. F.: Nitrogen Metabolism in the Sheep. Protein Digestion in the Rumen. Biochem. J., *64*, (1956): 705-714.

[3]Armstrong, D. G., and Blaxter, K. L.: The Heat Increment of Steam-Volatile Fatty Acids in Fasting sheep. Brit. J. Nutr., *11*, (1957): 247-272.

[4]Balch, D. A., and Rowland, S. J.: Volatile Fatty Acids and Lactic Acid in the Rumen of Dairy Cows

Receiving a Variety of Diets. Brit. J. Nutr., *11*, (1957): 288-298.

[6]Bienfet, V.: L'Acétonémie de la Vache Laitière. Ann. de Méd. Vet., *100*, (1956): 368-429.

[6]Black, A. L., and Kleiber, M.: The Tricarboxylic Acid Cycle as a Pathway for Transfer of Carbon from Acetate to Amino Acids in the Intact Cow. Biochim. et biophys. acta, *23*, (1957): 59-69.

[7]Clark, R., and Malan, J. R.: Alterations in the Blood Sugar and Ketone Levels Caused by Dosing Acetate, Propionate and Butyrate into the Rumen of the Sheep. Onderstepoort J. Vet. Res., *27*, (1956): 101-109.

[8]Elvehjem, C. A., and Krehl, W. A.: Dietary Interrelationships and Imbalances in Nutrition. Borden's Rev. Nutr. Res., *16*, (1955): 69-84.

[9]Gray, F. V., and Pilgrim, A. F.: Digestion of Nitrogenous Compounds by Ruminants. Nature, *178*, (1956): 94-95.

[10]Hundley, J. M.: In "The Vitamins," edited by W. H. Sebrell, Jr., and R. S. Harris. Vol. 2. Academic Press Inc., New York (1954): 551-565.

[11]Johnson, R. B.: A Theoretical Discussion of Ruminant Ketosis. Am. J. Vet. Res., *14*, (1953): 366-375.

[12]Johnson, R. B.: The Effects of the Administration of Acetic, Propionic, and n-Butyric Acids upon the Blood Glucose and Ketone Body Levels of Goats. Cornell Vet., *45*, (1955): 273-290.

[13]Johnson, R. B.: Ruminant Ketosis. Vet. Scope, *1*, (Winter, 1956): 2-10.

[14]Kleiber, M., Black, A. L., Brown, M. A., Luick, J., Baxter, C. F., and Tolbert, B. M.: Butyrate as a Precursor of Milk Constituents in the Intact Dairy Cow. J. Biol. Chem., *210*, (1954): 239-247.

[15]Kleiber, M., Smith, A. H., Black, A. L., Brown, M. A., and Tolbert, B. M.: Acetate as a Precursor of Milk Constituents in the Intact Dairy Cow. J. Biol. Chem., *197*, (1952): 371-379.

[16]Lewis, D., Hill, K. J., and Annison, E. F.: Studies on the Portal Blood of Sheep. 1. Absorption of Ammonia from the Rumen of the Sheep. Biochem. J., *66*, (1957): 587-592.

[17]Reid, R. L., Hogan, J. P., and Briggs, P. K.: The Effect of Diet on Individual Volatile Fatty Acids in the Rumen of Sheep, with Particular Reference to the Effect of Low Rumen pH and Adaptation on High-Starch Diets. Austral. J. Agric. Res., *8*, (1957): 691-710.

[18]Robertson, W. G., Lennon, H. D., Jr., Bailey, W. W., and Mixner, J. P.: Interrelationships Among Plasma 17-Hydroxycorticosteroid Levels, Plasma Protein-Bound Iodine Levels, and Ketosis in Dairy Cattle. J. Dai. Sci., *40*, (1957): 732-738.

[19]Rogers, T. A., and Kleiber, M.: The Biosynthesis of Milk-Fat in the Intact Cow. Biochim. et biophys. acta, *22*, (1956): 284-292.

[20]Schultz, L. H.: Use of Sodium Propionate in the Prevention of Ketosis in Dairy Cattle. J. Dai. Sci., *41*, (1958): 160-168.

[21]Schultz, L. H., and Smith, V. R.: Experimental Alteration of the Sugar and Ketone Levels of the Blood of Ruminants in Relation to Ketosis. J. Dai. Sci., *34*, (1951): 1191-1199.

[22]Shaw, J. C.: Bovine Ketosis. In "Advances in Veterinary Science," edited by C. A. Brandly and E. L. Jungherr. Vol. 2. Academic Press Inc., New York (1955): 262-306.

[23]Shaw, J. C.: Ketosis in Dairy Cattle. A Review. J. Dai. Sci., *39*, (1956): 402-434.

[24]Shaw, J. C.: Relation of Nutrition of the Rumen to Intermediary Metabolism of the Ruminant. Proc. Univ. of Maryand 1958 Nutrition Conference for Feed Manufacturers (March 20-21, 1958): 15-18.

[25]Shaw, J. C., and Lakshmanan, S.: Lactation and Hormones. In "Atomic Energy and Agriculture," edited by C. L. Comar. American Association for the Advancement of Science, Washington (1957): 305-334.

[26]Weinman, E. O., Strisower, E. H., and Chaikoff, I. L.: Conversion of Fatty Acids to Carbohydrate: Application of Isotopes to this Problem and Role of the Krebs Cycle as a Synthetic Pathway. Physiol. Rev., *37*, (1957): 252-272.

[27]Williams, R. J.: Biochemical Individuality. John Wiley and Sons, Inc., New York, 1956.

Study of Ketosis in a Dairy Herd

In an Aryshire herd, in Britain, in which ketosis had been an annual problem, the milk of each cow was tested for ketones (Rothera's test) weekly from November, 1954, to May, 1955. During that period, there were 4 clinical cases, 3 showing a severe decrease in milk yield and 1 showing nervous signs. There were also many subclinical cases.

On February 18, of 39 milk samples tested, 11 showed a high ketone and 9 a low ketone content; and on March 4, of 42 samples tested, 7 showed a high and 22 a low ketone content. The milk yield of some of these cows varied as much as 10 lb. per day. There was a sharp drop in milk ketones about two weeks after the silage ration was reduced by half and hay substituted on March 9. There was a further marked drop immediately after the cows were turned on grass on April 3.—*R. H. C. Penny in Vet. Rec. (Aug. 9, 1958): 641.*

Experimental Staphylococcic Mastitis.—Mastitis was produced in 11 of 12 goats inoculated in the teat canal with varying numbers of *Staphylococcus aureus* strain 201. Infection was followed by a systemic reaction and local mammary changes, varying from mild inflammation to rapidly developing gangrene. *Staphylococcus aureus* was never recovered from the spleens of goats which died but was recovered from one supramammary lymph node. Intramammary inoculations with sterile broth caused only minimal changes in the milk, while inoculation with heat-killed staphylococcic cultures also caused a transient systemic reaction.—*Vet. Bull. (Sept., 1958): Item 2717.*

Cutaneous Lesions on a Porpoise with Erysipelas

CHARLES F. SIMPSON, D.V.M., M.S.; F. G. WOOD, M.S.;
FRANKLIN YOUNG, M.Agr.

Gainesville, and Marineland, Florida

CUTANEOUS LESIONS occur in natural cases of erysipeloid in man and erysipelas in swine. This paper describes the gross and microscopic skin lesions on a porpoise that died of *Erysipelothrix rhusiopathiae* infection at Marineland Studios, Marineland, Fla.

On June 7, 1957, an 18-month-old porpoise (*Tursiops truncatus*), confined to feeding tanks, was observed to have well-defined cutaneous plaques on its ventrolateral side. No other signs of disease were noticed, but the porpoise died six days later, and a necropsy was conducted.

The porpoise was 187 cm. long, weighed 70 kg., had a subcutaneous fat thickness of 25 mm., and appeared to be in good condition. There were four sharply-defined square or rectangular skin plaques (fig. 1) on its left side, the largest being 35 mm. square and the smallest 18 by 25 mm. These lesions were oriented with the long axis of the body, were light gray, and were rough and slightly elevated as compared to the adjacent portion of normal skin which was smooth and of a silky texture. Immediately beneath the plaques, the subcutaneous fat was erythematous.

The only other gross lesions were splenomegalia, a yellowish liver, hemorrhagic gastritis, and the presence of Braunia sp. in the third compartment of the stomach.

BACTERIOLOGICAL AND PATHOLOGICAL FINDINGS

Spleen and lung tissues and subcutaneous erythematous fat immediately below the skin lesions were subjected to bacteriological examinations. Cultures were made on blood agar plates. Growth characteristics on all plates were identical and consisted of minute, colorless, translucent colonies. Organisms from these colonies were short, slender, gram-positive rods, sometimes curved. Acid, but not gas, was formed in lactose, glucose, galactose, and fructose. No fermentation occurred in starch, arabinose, mannitol, xylose, rhamnose, glycerol, salicin, maltose, trehalose, sucrose, sorbitol, adonitol, inulin, and raffinose. Hydrogen sulfide was produced and growth in gelatin stabs simulated a test tube brush.

Saline solution suspensions of spleen, subcutaneous fat, and organisms isolated from these two tissues were injected subcutaneously into white mice. The mice died 64 to 65 hours postinoculation and an organism, identical to that originally isolated from the porpoise, was recovered in pure culture and was identified as *Ery. rhusiopathiae*.[1]

It was found that *Ery. rhusiopathiae* could be grown readily in Brucella broth and on Brucella agar.* Moderate, uniform cloudiness without sedimentation was visible in broth after incubation at 37 C. for 24 hours. Small, translucent colonies, morphologically similar to those observed on blood agar plates, were seen on agar 24 hours following inoculation with a loopful of 24-hour broth culture.

Histological sections cut at 6μ were prepared from most organs and stained with hematoxylin and eosin. Fatty degeneration, bile duct hyperplasia, blood-filled sinusoids, and swollen Kupffer cells were observed in the liver. There was eosinophilic debris between the visceral and parietal layers of Bowman's capsules of kidney glomeruli.

Small, focal accumulations of lymphocytes and plasma cells occurred in the mucosa of the second, third, and fourth compartments of the stomach and small intestine. Neutrophils had infiltrated the stratified squamous epithelial lining of the first portion of the stomach. There was hemorrhage and lymphoid depletion of the mesenteric lymph gland.

The dermal papillae of the skin were thickened as a result of edema and infiltration by neutrophils. The superficial layers of the stratum corneum were separated by lenticular-shaped spaces filled with eosinophilic-stained fluid (fig. 2, 3). As a result, the affected area of skin projected above the adjacent normal portion. Blood vessels in the subcutaneous adipose tissue were congested.

Dr. Simpson and Mr. Young are with the Agricultural Experiment Stations, University of Florida, Gainesville; Mr. Wood is curator, Marineland Studios, Marineland, Fla.

Florida Agricultural Experiment Station journal series, No. 771.

*A product of Albimi Laboratories, Brooklyn, N. Y.

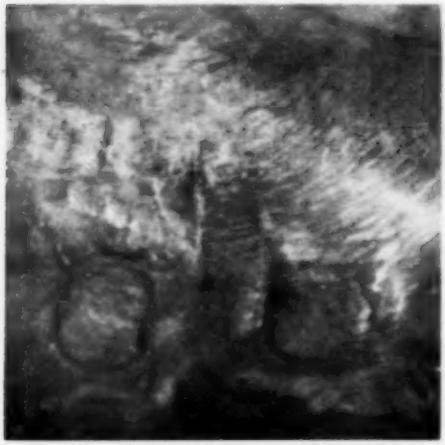


Fig. 1—Photograph of plaques on the skin of a porpoise with erysipelas.

Histological sections were stained also by the Gram technique. Organisms with typical morphology of *Ery. rhusiopathiae* were seen in the dermal papillae of affected skin, sinusoids of liver, mesenteric lymph gland, spleen, and the mucosa of the first, second, and third compartments of the stomach.

DISCUSSION

During the past six years, at least 8 porpoises believed to have been infected with erysipelas at the Marineland Studios have had lesions on the skin in the form of sharply-outlined, elevated plaques.

In addition to the animal described here, 2 others died in the past six years following the development of skin lesions. One, which was 9½ months old, showed skin lesions five days prior to death. Bacteriological and histological data are not available, but necropsy records indicate that the only gross pathological findings were splenomegalia, skin plaques, and erythematous areas beneath the skin lesions. Treatment was not attempted. The other porpoise died of a bacteremia five days after the development of skin lesions. Vitamin A was administered, without effect.

Five porpoises which had developed cutaneous plaques recovered within two and one half to three weeks. Two of these were treated with vitamin A and a third recovered without treatment. Two animals were treated with penicillin and vitamin B com-

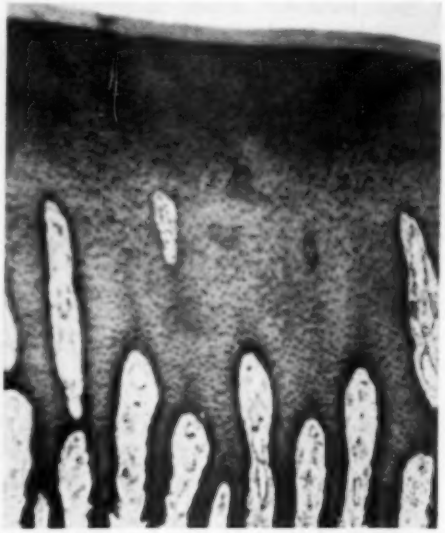


Fig. 2—Photomicrograph of normal porpoise skin. H & E stain; x 45.

plex parenterally and they responded to treatment.

It was reported[2] that the acute form of erysipelas in porpoises could terminate in death 20 minutes to 36 hours following the first indication of sickness. Skin plaques were found in none of these cases.[3]

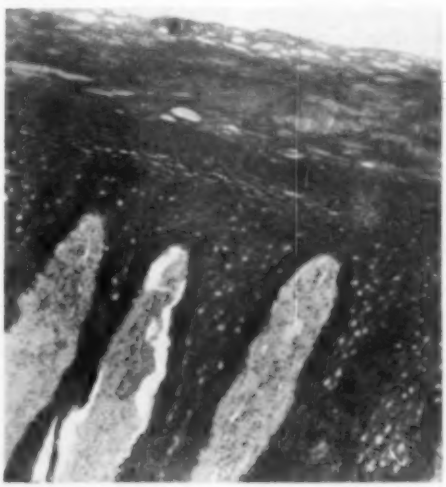


Fig. 3—Photomicrograph of a plaque on the skin of a porpoise. Notice thickened dermal papillae due to edema and cellular infiltration. H & E stain; x 64.

Four additional deaths in porpoises at this studio, due to the acute stage of erysipelas, have been diagnosed since this original report.

SUMMARY

1) Erysipelas was diagnosed in a porpoise by histological and bacteriological techniques. Square or rectangular plaques on the skin were associated with the disease in this porpoise.

2) It was found that Brucella broth and agar supported the growth of *Erysipelothrix rhusiopathiae*.

References

[1]Breed, R. S., Murray, E. G. D., and Hitchens, A. P.: Bergey's Manual of Determinative Bacteriology. 6th ed. Williams and Wilkins Co., Baltimore (1948): 410-411.

[2]Seibold, H. R., and Neal, J. E.: Erysipelothrix Septicemia in the Porpoise. J.A.V.M.A., *128*, (June 1, 1956): 537-539.

[3]Wood, F. F., curator, Marineland Studios, Marineland, Fla.: Personal communication, Sept., 1957.

Two Cases of *Erysipelothrix Rhusiopathiae* Infection in Chickens

JOHN G. KILIAN, D.V.M.; W. E. BABCOCK, D.V.M., M.Sc.; E. M. DICKINSON, D.V.M., M.Sc.

Corvallis, Oregon

A REVIEW of the literature confirms that a natural infection of *Erysipelothrix rhusiopathiae* rarely occurs in chickens in the United States. One case of natural infection, found in conjunction with fowl cholera, was described in Nebraska in 1943.[1] Two cases were reported in 1953;[2] 1 existed concurrently with lymphomatosis and the other where chickens and pigs were allowed to mingle. Other recent cases[3,4] indicate there may be an increase of this infection in chickens.

Several attempts at this laboratory to artificially establish the infection in chickens by various routes of inoculation were unsuccessful.

CASE REPORTS

This report deals with the first isolations of *Ery. rhusiopathiae* from chickens in Oregon with natural infections. Two unusual cases were encountered; 1 in broilers and the other, which persisted over a period of several months, in a laying flock.

Case 1—Broilers.—This case involved a flock of 2,500 White Rock chickens, 6 weeks old, that were to be marketed as broilers at about 10 weeks of age. They had been purchased from a local hatchery and were part of a continuous broiler-producing operation that receives and also markets about 2,500 birds weekly.

From the Department of Veterinary Medicine, Oregon State College Agricultural Experiment Station, Corvallis.

The brooder house and most equipment were new at the time of housing. The birds were confined to the house on fresh, fir wood shavings; a wooden watering trough in the middle of the house had collected a thick layer of mold in the bottom. The litter became damp, which was unusual for this time of year, but mortality during the first few weeks was low. Sparrows entered the house at various times and there was a constant problem of rats in the feed room. Other broilers which were housed about 25 ft. away did not become infected.

On Oct. 28, 1955, when 6 weeks old, 4 birds—3 alive and 1 dead—were presented to the laboratory for necropsy. About 200 had died recently and about 200 were sick. They had been on a starter feed containing a small amount of a broad-spectrum antibiotic. The owner suspected coccidiosis because of the appearance of the chicks at 5 weeks of age, and had given them 0.04 per cent sulfaquinoxaline in the drinking water, with negative results.

At necropsy, a severe nephritis with urate deposits, and ulceration of both the proventriculus and ventriculus, were gross lesions common to all birds. Inoculum from the liver and heart blood was placed on a medium consisting of 31 Gm. of nutrient agar, 20 Gm. of gelatin, and 15 Gm. of peptone in 1,000 ml. of distilled water. The tentative diagnosis was nonspecific nephritis and it was recommended that the level of oxytetracycline in the feed be increased

to 200 Gm. per ton. Further, the water and feed troughs were to be cleaned and disinfected and fresh, clean litter supplied.

The culture medium had bacterial growth, after 24 hours' incubation, characteristic of *Ery. rhusiopathiae* and some of this growth was transferred to thioglycollate broth (Difco). The organisms stained as small, gram-positive, pleomorphic, granulated rods.

The standard mouse protection test was used to identify the organism. Using 4 white mice, 2 were protected with 0.5 ml. anti-swine erysipelas serum given subcutaneously, then all 4 mice were given 0.2 ml. subcutaneously of the inoculated thioglycollate broth after 24 hours' incubation. The 2 unprotected mice died in 96 hours and *Ery. rhusiopathiae* was recovered. The protected mice survived the challenge inoculation.

At the time the owner was notified of the diagnosis of erysipelas, it was learned that the birds were improved and losses had stopped. Because of the economics involved, the only treatment was the continued high-level antibiotic feeding until the birds were sent to market.

The birds varied greatly in size (av. 2 lb. 7 oz.) when processed at 9 weeks and 3 days of age. The owner estimated a loss of 300 birds from all causes.

Case 2—Laying Pullets.—The owner of a flock of 800 White Leghorn pullets, 9 months old, brought 2 birds to the laboratory for examination on Dec. 15, 1955. He stated that his flock was divided, with about 400 birds in each of two houses. In the house from which the birds examined came, 12 had died suddenly and several others were sick. Feed consumption was normal and egg production was well over 70 per cent. At necropsy, both birds had a moderately severe nephritis. In addition, 1 bird had a generalized peritonitis, the result of egg material in the abdominal cavity, which made it unsuitable for bacteriological examination. The second bird had hepatitis, a swollen spleen, severe ascariasis, and ulceration in the proventriculus and ventriculus.

Inoculum from the liver and spleen of the second bird was placed on the fortified nutrient agar culture medium previously described. It was recommended that the remaining birds be treated with a piperazine preparation, that the equipment be cleaned, and that clean litter be supplied.

The culture medium had bacterial growth after 24 hours' incubation, characteristic of *Ery. rhusiopathiae*, and some of this growth was transferred to thioglycollate broth. After 24 hours' incubation, the broth was used in a standard mouse-protection test for erysipelas, as previously described. This test was positive for erysipelas and the organisms from the broth stained as small, gram-positive, pleomorphic, granulated rods.

Upon reporting the erysipelas diagnosis to the owner five days after the necropsy, it was learned the piperazine treatment had yielded good results and mortalities had ceased, although egg production remained at about 50 per cent. The owner had cleaned the house and equipment and had started feeding a ration containing 100 Gm. of oxytetracycline per ton.

These birds had been purchased from a local hatchery, as day-old chicks, and had been brooded and ranged in houses and on soil where several apparently healthy ewes and lambs had wintered. While on range, the birds had free access to the laying houses which had been idle for two years previously. A ration containing a coccidiostat was fed from 4 weeks of age until they were housed at 6 months of age. The entire mortality then had been less than 3 per cent from all causes. Before occupancy, both the brooder and laying houses were cleaned and disinfected with lye solution. Soon after the birds were housed, egg production rose to over 70 per cent. They maintained this level of production until early December, when it started dropping in one house. A few (about 6) soft-shelled eggs were found on the floor of this house daily (this persisted until March).

The next contact with the flock was on Jan. 4, 1956, when the owner presented 2 birds for necropsy. He reported that 38 had died suddenly, several having been found dead on the roost, and he estimated that 15 more sick birds would probably die.

At necropsy, 1 bird had an enlarged spleen and the liver was green from bile retention. The second bird showed infarcts in the heart and spleen, also severe hepatitis with areas of necrosis in the liver. Inoculum from the liver, spleen, and heart blood from both birds placed in tubes of previously described medium yielded bacterial growth characteristic of *Ery. rhusiopathiae*. Mouse-protection tests conducted

with the organism again were positive for erysipelas.

When notified of the diagnosis, the owner started supplemental feeding of 45 to 50 lb. of medicated mash, containing 100 Gm. of oxytetracycline per ton, every other day. This amounted to a little over one half the daily feed requirement for the pen of 400 pullets.

On March 13, another bird submitted for necropsy showed lesions in the spleen and liver suggestive of erysipelas. Inoculum from these organs, placed on previously described medium, yielded typical *Ery. rhusiopathiae* organisms that were positive also on mouse-protection tests. The owner reported that since the January 4 visit, a total of 15 birds had died, that the appearance of the flock had improved, and that egg production had risen above 50 per cent.

No further losses occurred and egg production was well over 50 per cent when the flock was marketed on April 4. After the March 13 visit, the only medication was the intermittent supplemental feeding of antibiotic previously described. Although there was over 16 per cent mortality during the four-month observation period, the disease did not spread to the other nearby house of 400 pullets on the same farm.

SUMMARY

1) Two cases of natural *Erysipelothrix rhusiopathiae* infection, 1 in broilers and 1 in pullets, are reported.

2) The source of infection was not determined in either case.

3) The disease did not appear to spread to chickens in other houses on the same farm.

References

[1]Breed, F.: *Erysipelothrix Rhusiopathiae* and *Pasteurella Avicida* in Chickens. Vet. Med., *38*, (1943): 431.

[2]Evans, W., and Narotsky, S.: Two Field Cases of *Erysipelothrix Rhusiopathiae* Infection in Chickens. Cornell Vet., *44*, (1954): 32-35.

[3]Vance, H. N., and Whenham, G. R.: An Erysipelas Outbreak in Chickens. Canad. J. Comp. Med., *22*, (1958): 86-87.

[4]Vickers, C. L., and Bierer, Bert W.: Erysipelas in a Flock of Chickens. J.A.V.M.A., *133*, (Aug. 15, 1958): 223-224.

Erysipelas in Mink.—Erysipelothrix rhusiopathiae was isolated from 1 of 3 mink which died of erysipelas.—*Med. Wtryn. (March, 1958): 142.*

Erysipelas Bacillus Distribution

Erysipelothrix rhusiopathiae has been found either as a pathogen or a saprophyte in swine, sheep, cattle, horses, dogs, wild boars, reindeer, kangaroos, guinea pigs, mice, wild rodents, turkeys, chickens, ducks, geese, guinea fowl, pigeons, sparrows, eagles, seals, porpoise (dolphin), fish (both salt and fresh water), shells, and man.

It is also found in animal cadavers and decaying plants. As a saprophyte in healthy animals, it is found mainly in the tonsils and Peyer's patches.—*H. R. Ehrsam in Schweiz. Archiv. fur Tierkeilk. (April, 1958): 202-208.*

Pathogenesis and Growth of Erysipelas Bacteria in Fish.—The *Bacterium munsepticum* which causes erysipelas in swine and erysipeloid in man, the disease called "fischrose" (fish-rose) by fishermen is not pathogenic for fish but it has been cultured, even during the cold seasons, from ten species of salt-water fish and from freshwater fish and remains pathogenic for swine and mice.—*G. Wellman in Abhandlungen aus der Fischerie (1950): 489-504.*

[Dr. Wellman writes that "We do not know if erysipelas organisms are already present on live fish or reach them after the catch (June 16, 1958).—ED.]

Spontaneous Erysipelas in a Herd of Research Swine.—Spontaneous erysipelas has been studied, since 1952, in a herd of swine selected for that purpose in Germany. The only ones which became ill had little or no serologically identifiable immunity to erysipelas. When exposed, they developed variable degrees of the disease, from subclinical to peracute septicemia. One with a culturally demonstrated bacteriemia showed only a short illness and no skin lesions. Some pigs nursing affected sows showed no illness although some of them had subclinical infection.—*G. Wellmann in Arch. f. exptl. Vet.-med., 12, (1958): 62-67.*

Intracutaneous Tests for Brucellosis in Swine.—Two Brucella allergens were used experimentally in healthy swine and in those artificially infected with *Br. suis*. Both produced specific and distinct reactions in the infected animals.—*K. Dedić, et al. in Arch. f. exptl. Vet.-med., 2, (1958): 193.*

Steatitis ("Yellow Fat") in Cats Fed Canned Red Tuna

T. O. MUNSON, V.M.D.; J. HOLZWORTH, D.V.M.;
E. SMALL, D.V.M.; S. WITZEL, D.V.M.;
T. C. JONES, D.V.M.; H. LUGINBÜHL, Dr. med. vet.

Boston, Massachusetts

YELLOW DISCOLORATION of fat associated with deposition of an acid-fast pigment was first reported in experimental chicks and rats fed diets high in unsaturated fatty acids and low in vitamin E.[5,8] A similar or identical disease condition also occurs in mink,[4,7,9] swine,[6] and kittens[1,2] reared on rations high in fish scrap.

Of the kittens, 2 were 7-month-old tabbies, male and female litter mates raised from weaning exclusively on a fish-base commercial canned cat food.[1] The 2 others, owned by different persons, were 6-month-old Siamese, a male and female, likewise fed from weaning on a canned cat food high in fish, but supplemented in varying degree with horsemeat and milk.[2]

It is believed that the presence in a ration of large amounts of unsaturated fatty acids together with a deficiency of the antioxidant, vitamin E, results in deposition of nonsoluble fatty-acid fractions. In experimental studies[2,6,7] with cats, swine, and mink, development of this pigment, often called "ceroid" because of its waxy nature, was inhibited by feeding of vitamin E (in the case of kittens, a minimum of 20 mg. *alpha* tocopherol a day).

Recently, steatitis has been observed at the Angell Memorial Hospital in 4 cats, from different environments, but in the diets of which the common denominator was a commercial canned cat food consisting entirely of red tuna.*

The purpose of this report is to draw attention to the easily recognizable signs of the condition, indicate means of accurate diagnosis, and make suggestions as to treatment.

CASE REPORTS

Case 1.—A plump and glossy-coated spayed female tabby cat that lived entirely indoors was admitted to the hospital on Feb. 25, 1958. This and another female, not a litter mate, had been acquired in Washington, D.C., in April, 1957, at the age of 6 weeks. They were given the red tuna product, horsemeat, and a canned dog food. The first one ate the red tuna almost exclusively and no milk; the other kitten ate the three foods equally and also drank about a quart of milk a week.

When the owner moved to Boston in October, she was unable to obtain the red tuna for about a month and the first cat merely picked at other things and drank a little milk. When the red tuna again became available, she ate it exclusively as before.

For a day and a half before admission to the hospital, the cat was listless and moved with reluctance and difficulty, her hindlegs, particularly, being affected. On examination, she seemed to be unusually "solid," but could scarcely be handled because of extreme generalized tenderness. Her abdomen was so tense and sensitive that it could not be palpated, and even a light touch over her back caused her to lash out in pain. Her temperature was 104.6 F. The total leukocyte count was 42,600 per cubic millimeter, with a marked neutrophilia.

The cat was treated first with a penicillin-streptomycin mixture and then, as her temperature remained high, chloramphenicol. Although she did not eat or drink, there was no significant dehydration or loss of weight. Four days later, the leukocyte count was 52,400/cmm. (segmented neutrophils 76%, band neutrophils 10%, metamyelocytes 4%, eosinophils 1%, and lymphocytes 9%).

By the eighth day, although the leukocyte count had risen to 67,100, the cat seemed much less tender, her temperature was normal, and she had nibbled at some of the red tuna. She was therefore discharged. For two days at home, she seemed to be returning to normal but then relapsed into inappetence and inactivity.

Drs. Munson and Holzworth are clinical staff members, Drs. Small and Witzel are interns, Dr. Jones is pathologist, and Dr. Luginbühl is resident in pathology at the Angell Memorial Animal Hospital, Boston, Mass.

*The authors will furnish the brand name of the food on request.

Fig. 1—Thickened, nodular yellow fat in the mesentery (case 1). The dark spots seen in this black and white photograph were bright yellow in the subject, and the intervening areas were yellowish tan.

On readmission to the hospital on March 10, her temperature was 103.6 F., and all over her body, especially in the axillae, groin, and perineum, the subcutaneous tissue felt lumpy. The cat did not seem as seriously affected as on the first admission, but her leukocyte count was 71,300/cmm. (segmented neutrophils 79%, band neutrophils 17%, metamyelocytes 2%, eosinophils 2%). She ate a little red tuna that afternoon but was found dead early the following morning.

At necropsy, the gross lesions were confined to the adipose tissue in the subcutis and the abdominal and thoracic cavities. The panniculus adiposus was 1.5 cm. thick over the lateral thorax and 3.0 cm. over the abdomen, with a yellowish-tan color and granular, lobulated appearance. These changes affected all the body fat except that of the head and neck. A sharp line of demarcation at the shoulder was seen between the thin layer of white fat over the cervical region and the yellow, coarsely nodular fat over the rest of the body.

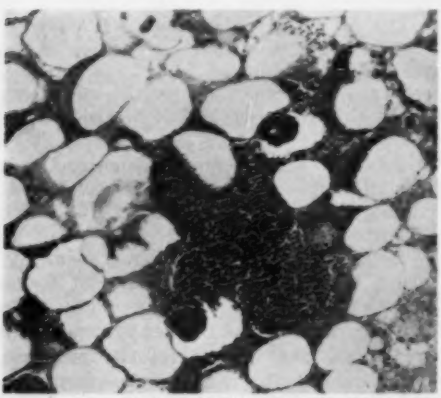


Fig. 2—Intense leukocytic infiltration of subcutaneous fat (case 1). Dark cells in masses between fat cells are neutrophils; many are necrotic. H & E stain; x 110.

The omentum and mesentery had a thick, almost leathery appearance, and their fat content was yellowish tan with points of bright yellow (fig. 1). At its attachment to the intestine, the fat-thickened mesentery was deflected in a sharp fold (fig. 1). The peritoneal cavity contained about 10 ml. of yellow-tinged fluid.

Microscopic examination revealed severe inflammatory changes in all sections of adipose tissue. The dominant feature was the infiltration of neutrophils into irregularly sized areas between the large globular fat cells. These leukocytes among the clear globules of adipose tissue formed solid masses and radiated out between the fat cells. In many areas, lymphocytes, plasma cells, and macrophages were evident in the stroma between fat cells, but were greatly outnumbered by the neutrophils (fig. 2). Amorphous material, of eosinophilic staining character, could be detected in the areas of inflammation between the cells and

sometimes inside the cells. In sections stained with Ziehl-Neelsen's method, this material was acid-fast and was seen as bright red substance deposited around the periphery of the fat globules and between the fat cells.

Tiny, sharply outlined, spherical globules, yellowish and acid-fast, were also scattered through the sections, particularly in the stroma between fat cells. They were sometimes irregularly shaped and of various sizes, but rarely were more than one fifth the diameter of a fat cell.

Case 2.—A spayed female tricolor tabby cat, 2 years old and in good flesh, was admitted to the hospital on April 5. For three days, she had not played or jumped as usual and cried out in pain when handled. She was thirsty but would not eat. For the previous six or seven weeks, her diet had been mainly red tuna, supplemented occasionally by boiled liver; no milk was given. The cat had freedom to run outside, but was mostly indoors.

When examined, she was reluctant to move and showed pain, particularly when touched over the back, sternum, and abdomen. The subcutis of the ventral thorax and abdomen felt lumpy. The cat's temperature was 102.6 F., and the total leukocyte count was 24,650/cmm. (segmented neutrophils 70%, band neutrophils 8%, metamyelocytes 2%, and lymphocytes 20%). A diagnosis of nutritional steatitis was made and appeared to be confirmed when a small incision into the subcutis over the sternum disclosed grayish lumpy fat.

Treatment consisted of 54 mg. of d, *alpha* tocopherol (Tocopherex*) daily, a multivitamin preparation, and penicillin-streptomycin. During 24 days of hospitalization, the cat would eat horsemeat and milk sporadically, the marked sensitivity to palpation and the lumpiness of the subcutaneous fat continued, and her temperature ranged mostly between 102.6 and 103.4 F., dropping to normal for only one two-day period.

On the seventh day, the total leukocyte count was 40,000/cmm. and on the sixteenth, 35,530/cmm. On the twenty-fourth day, because of severe weight loss and persistent fever and soreness, the owner had the cat destroyed.

The gross findings at necropsy were nearly identical to those in the first cat. The subcutaneous fat was deep yellow, nodular, and 0.5 to 1.0 cm. thick over the thorax and abdomen. The subcutis of the neck and head had a thin layer of normal white fat with an abrupt change to yellow fat at the level of the shoulder. The mesenteric and omental fat was brownish yellow with foci of bright yellow and was obviously thicker than normal. The pericardial fat was not excessive and for the most part was white, but some tan foci were scattered through it. No other gross lesion was seen.

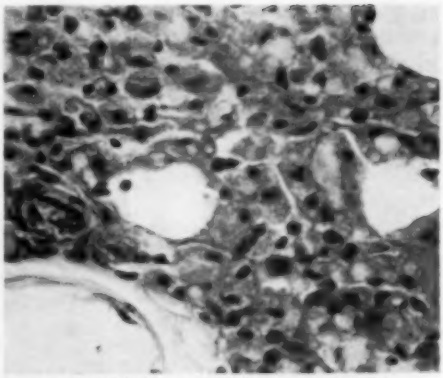


Fig. 3—Lipid-laden macrophages in subcutaneous adipose tissue (case 2). Yellowish to brown pigment filled the cytoplasm of most of the cells in this area. H & E stain; x 400.

The microscopic findings were also quite similar to those in the first cat. In large areas, the adipose tissue was infiltrated with neutrophils and necrotic debris. Canary-yellow pigment, indistinguishable from that of the first case, was evident, and its acid-fast nature was readily demonstrated. In addition, small islands of epithelioid cells, many laden with brown, tan, or yellow pigment, could be found scattered among the fat cells (fig. 3). In a few of these areas, foreign-body giant cells of the Langhans' type could be distinguished. The tan foci in the pericardial fat proved to be small areas of steatitis identical to those in other adipose tissues.

Case 3.—A 10-month-old female tabby cat, spayed two months before, entered the hospital on May 5 because of soreness, inactivity, and depression increasing over a

*Tocopherex is produced by E. R. Squibb & Sons, New York, N. Y. Each capsule contains 18.5 mg. of d, alpha tocopheryl acetate with associated mixed tocopheryl acetates, equivalent to 27 mg. of d, alpha tocopherol, or 25 I.U. vitamin E.

five-day period. She would no longer jump onto chairs, and her appetite had diminished. The cat never went out of doors, so there was little likelihood of an injury. Her diet was varied, consisting of cooked and raw fish, raw beef and liver, and canned red tuna cat food (about one can a week for three months.)

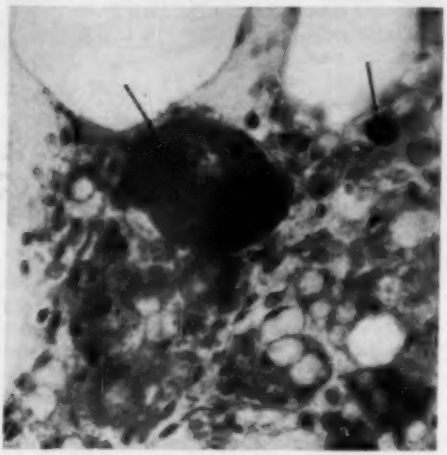


Fig. 4—Globules (arrows) of acid-fast pigment in macrophages between fat cells in the panniculus adiposus (case 4). Similar but less dense pigment is seen in the cytoplasm of macrophages. Ziehl-Neelsens' acid-fast stain; x 540.

When examined initially, her temperature was 103.7 and she had tender masses in the groin, which suggested hernia. However, on the basis of diet and generalized sensitivity, especially over the back and chest, a diagnosis of nutritional steatitis was made. The owner would not consent to a biopsy.

On admission, the leukocyte count was 12,500/cmm. (segmented neutrophils 42%, band neutrophils 30%, eosinophils 8%, and lymphocytes 20%). Treatment consisted solely of 54 mg. daily of d, *alpha* tocopherol, and the owner was encouraged to spend time with the cat in order to get it to eat adequate amounts of a variety of meats.

After several days, the cat's temperature returned to normal but, as the tenderness persisted and the leukocyte count rose to 23,650/cmm., with the eosinophils remaining elevated (9%), cortisone was started on the tenth day. Five days later, the cat was much improved and the leukocyte count had dropped to 18,000/cmm. From then on, the soreness subsided slowly but steadily, leukocyte counts on the twenty-third and twenty-ninth days were 15,000/cmm., and on the thirtieth day the cat was discharged.

Case 4.—A spayed female tabby cat, 1½ years old, in moderately good flesh, was admitted on June 12 because of lameness, lethargy, and inappetence increasing over a week's time. She had lost her agility and had difficulty jumping onto chairs. Until April, she had eaten a commercial dog food but without enthusiasm. With the feeding of red tuna, her appetite had increased remarkably, and for two months she had eaten one can a day, together with occasional meat scraps.

The cat's temperature was 103.6 F. Besides generalized shedding and some areas of punctate reddening in the mouth behind the last lower molars, there was extreme sensitivity to handling, especially over the back. The leukocyte count was 22,250/cmm. (segmented neutrophils 37%, band neutrophils 25%, metamyelocytes 4%, eosinophils 3%, and lymphocytes 31%). A diagnosis of nutritional steatitis was made, and a biopsy of subcutaneous fat was taken from the sternal region.

The biopsy specimen consisted of a thin segment of subcutaneous fat which was solid, yellowish tan, and nodular. Microscopically, there were definite inflammatory changes in the panniculus adiposus. Globules of yellowish to tan, acid-fast pigment were evident between the fat cells and in macrophages (fig. 4). Neutrophils were seen in small groups, but none was obviously necrotic.

Epithelioid cells, laden with yellow to brown pigment, were most numerous in the solid islands of cells which infiltrated and displaced fat cells. Langhans'-type giant cells were relatively numerous (fig. 5). The preponderance of these epithelioid elements was interpreted to indicate that the disease was less fulminant and perhaps of longer duration than in cats 1 and 2. The tissue reaction, however, was of similar nature in all 3 cases.

Treatment included 27 mg. of d, *alpha* tocopherol daily, penicillin-streptomycin, B complex, and a varied meat diet. The cat's temperature ranged between 103.6 and 104.0 F. till the fourth day, when it dropped to normal and there was a notice-

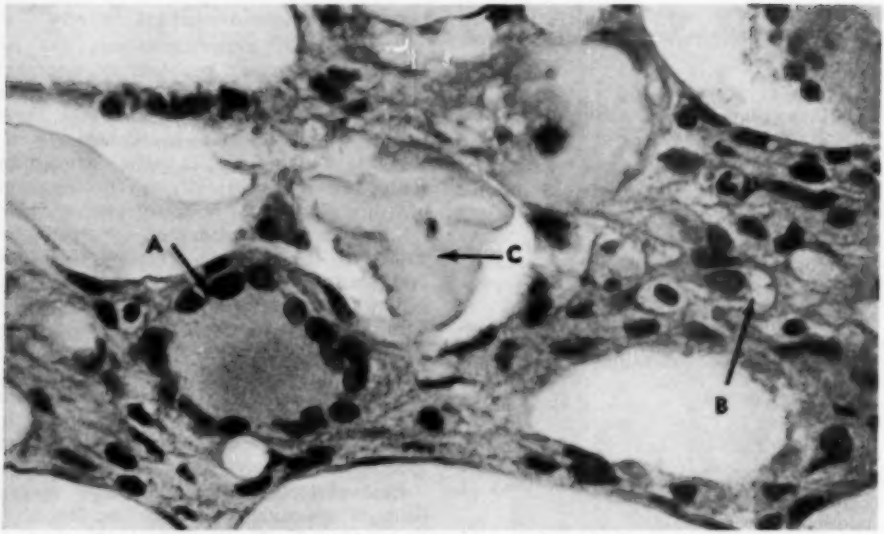


Fig. 5—Giant cell (A) and epithelioid cells (B) between fat cells (case 4). Notice irregularly shaped material in fat globules (C). H & E stain; x 800.

able decrease in sensitivity. The cat was discharged on the eighth day, greatly improved, with 27 mg. of tocopherol and 1 mg. of cortisone (Delta-Cortef**) to be given daily for ten days. The leukocyte count at that time was 19,100/cmm. (segmented neutrophils 46%, band neutrophils 14%, metamyelocytes 1%, eosinophils 3%, and lymphocytes 36%).

DISCUSSION AND CONCLUSION

From the 4 cases of steatitis described, a clearly recognizable clinical picture emerges, in which decreased agility, extraordinary tenderness, lumpiness of the subcutaneous fat, and fever are the outstanding signs. By what may or may not be coincidence, all 4 cats were spayed female tabbies, and were young (10 months to 2 years).

The characteristic laboratory finding is an elevated leukocyte count, with a neutrophilia and varying degrees of eosinophilia. Positive confirmation of the diagnosis of steatitis is readily obtained through a biopsy.

The prognosis would seem to be good if the condition is recognized promptly and the cat receives specific treatment. In our limited experience, it appears that a minimum of 27 mg. of d, *alpha* tocopherol a day will bring about improvement, provided also that the patient can be dissuaded from the red tuna and induced to eat a variety of other foods.

In view of the increase in eosinophils conspicuous in cat 3 and probable in 1 and 4, it is suggested that cortisone may perhaps hasten clinical improvement by combating the intense inflammation associated with the foreign-body reaction in the fat.

Although antibiotics were given in three instances, they may have been unnecessary. Because of the painful and inflamed condition of the subcutaneous tissue, it would seem inadvisable to resort to subcutaneous administration of fluids as supportive treatment.

As to the cause, it can only be stated at this time that the common factor in all 4 cases was the feeding of a canned red tuna cat food—in two instances as practically the whole ration, and in the other two as perhaps less than half. In view of experimental evidence,[2] which points toward fish products as the cause of steatitis, the tuna fish diet appears to be involved in these cases. Whether there is an intrinsic deficiency in this type of food or whether its

**Delta-Cortef is produced by the Upjohn Co., Kalamazoo, Mich.

harmfulness is due to deterioration in processing or storage remains to be determined.

References

[1]Coffin, D. L., and Holzworth, J.: "Yellow Fat" in Two Laboratory Cats: Acid-Fast Pigmentation Associated with a Fish-Base Ration. Cornell Vet., *44*, (1954): 63-71.

[2]Cordy, D. R., and Stillinger, C. J.: Steatitis ("Yellow Fat Disease") in Kittens. North Am. Vet., *34*, (1953): 714-716.

[3]Cordy, D. R.: Experimental Production of Steatitis (Yellow Fat Disease) in Kittens Fed a Commercial Canned Cat Food and Prevention of the Condition by Vitamin E. Cornell Vet., *44*, (1954): 310-318.

[4]Dalgaard-Mikkelsen, S., Kvorning, S. A., Momberg-Jorgensen, H. C., Petersen, F. H., and Schambye, P.: Studier over "gult fedt" hos mink. Nord. Vet.-med., *5*, (1953): 79-97.

[5]Dam, H.: Studies on Vitamin E Deficiency in Chicks. J. Nutr., *27*, (1944): 193-211.

[6]Davis, C. L., and Gorham, J. R.: The Pathology of Experimental and Natural Cases of "Yellow Fat" Disease in Swine. Am. J. Vet. Res., *15*, (1954): 55-59.

[7]Gorham, J. R., Baker, G. A., and Boe, N.: Observations on the Etiology of Yellow Fat Disease in Mink: Preliminary Report. Vet. Med., *46*, (1951): 100-102.

[8]Mason, K. E., Dam, H., and Granados, H.: Histological Changes in Adipose Tissue of Rats Fed a Vitamin E Deficient Diet High in Cod Liver Oil. Anat. Rec., *94*, (1946): 265-288.

[9]Mason, K. E., and Hartsough, G. R.: "Steatitis" or "Yellow Fat" in Mink, and Its Relation to Dietary Fats and Inadequacy of Vitamin E. J.A.V.M.A., *119*, (July, 1951): 72-75.

Selenium and Muscular Dystrophy

A deficiency of vitamin E and associated tocopherols is apparently not the only factor in causing nutritional muscular dystrophy in lambs. In several trials, none of the lambs from ewes fed, during pregnancy, a ration which contained wheat bran and linseed oil meal developed this stiffness, whereas 20 to 60 per cent of the lambs on the same roughage plus raw beans or raw beans and DPPD showed signs of the disease.

In the experiment, completed in 1958, 8 of 18 (44%) in the control group developed the disease, whereas only 1 of 21 lambs (5%) from ewes fed the basal ration plus 1 p.p.m. of selenium was affected. The linseed oil meal contained 1.1 μg./Gm. (1.1 p.p.m.) of selenium, whereas the roughage and raw beans contained only 0.012 to 0.08 p.p.m. of selenium.

Selenium is apparently one of the factors effective in preventing muscular dystrophy in lambs.—*D. E. Hogue, Cornell Nutr. Conf., Nov. 12-14, 1958.*

Hypomagnesemia—Grass Tetany

Stating that hypomagnesemia has become an important problem in Denmark only during recent years, although first reported in 1931, the author lists three factors which appear to be etiologically important. These are: (1) the magnesium content of the feed; (2) the protein, and possibly potassium, in the grass; and (3) the influence of weather on serum-magnesium and the possible presence of serum-magnesium regulators.

The condition is treated by intravenous administration of calcium and magnesium chlorides or by subcutaneous injection of 100 to 200 ml. of a 25 per cent solution of magnesium sulfate, possibly with calcium borogluconate. Relapse is prevented by oral administration of 50 Gm. of magnesium oxide per day for two to three weeks.

Cost of treating the fields with magnesium is so high as to be impractical in Denmark.—*M. G. Simesen in Nord. Vet.-med., 9, (April, 1957): 307.*—R. KLUSSENDORF.

Feeding Enzymes to Poultry.—Since the addition of pepsin and other proteolytic enzymes to certain rations improved the growth of pigs up to 5 weeks of age, similar methods of feeding were tried with broilers, growing turkeys, and laying hens. There was a marked improvement in growth and feed utilization in the younger birds.—*L. S. Jensen, Cornell Nutr. Conf., Nov. 12-14, 1958.*

Effect of Antibiotics on Growth of Rhesus Monkeys.—In a series of experiments, Rhesus monkeys given chlortetracycline or oxytetracycline at a level of 70 to 140 mg. per liter of drinking water, for 12 to 14 days, gained weight at a significantly greater rate than did untreated controls. There were no apparent ill effects.—*C. R. Coid in Vet. Rec. (Sept. 6, 1958): 726.*

Porcine Parakeratosis in South Australia.—Parakeratosis was reported in swine in South Australia. It responded rapidly to low levels of zinc supplementation. Improvement was enhanced by housing the pigs. Some pigs which showed no signs of parakeratosis made a marked growth response to the zinc treatment.—*W. J. Wilkie et al. in Austral. Vet. J. (June, 1958): 172.*

Dr. Hay Appointed to Central Office Staff

The appointment of Dr. James R. Hay as director of professional relations and membership services for the AVMA, effective Nov. 17, 1958, has been announced by the Executive Board.

Dr. Hay is assuming a new position in the Association. He will serve as a field secretary for the Councils on Education, Veterinary Services, and Public Health and Regulatory Veterinary Medicine. He will be concerned with intraprofessional relations, internal public relations, and will maintain an active liaison with allied organizations. He will also endeavor to enhance the promotion of student AVMA chapters, will improve and enlarge the central office contact with constituent associations, and will promote the expansion of the AVMA film library.

Dr. Hay was born in London, Ont., Canada, on Dec. 30, 1912, but spent most of his boyhood on a farm in Maryland. He attained the B.A. degree at Ohio State University in 1934 with a major in bacteriology. During the following year, he was employed by the then BAI to conduct brucellosis tests at the Reynoldsburg, Ohio, laboratory. Following graduation, in 1939, from the veterinary college at Ohio State University, he was employed by the University of Delaware to work in their mastitis disease research program and remained there for three years. Then, from 1942 to 1944, he was associated with Corn States Serum Co., in their diagnostic laboratory and in biological production.

In 1944, he joined the U. S. Navy, and was assigned to communications and personnel activities. Part of his Naval service was in the Philippines and he attained the grade of lieutenant. After his separation from military service in 1946, he engaged in a general practice at Chillicothe, Ohio, and remained there until he was appointed state veterinarian in 1952. It was during his tenure of office that the first state diagnostic laboratory was established and that an animal disease reporting system was instituted in Ohio. In 1957, the governor appointed him to the post of director of the Ohio Department of Agriculture.

Dr. Hay married the former Thelma Simms, of Columbus, Ohio, in 1939, and they now have one daughter, Linda, age 13.

Dr. James R. Hay

He has been a member of the AVMA since graduation, is the incumbent first vice-president of the United States Livestock Sanitary Association, secretary-treasurer of the North Central Association of the State Departments of Agriculture, and is a past exalted ruler of the Benevolent and Protective Order of Elks. He is also a member of the Ohio State Veterinary Medical Association, National Association of State Departments of Agriculture, Phi Zeta, Alpha Psi, and Masonic bodies including the Shrine.

Dr. Hay's wide background of training and experience, his many and varied contacts with agricultural organizations, and his natural aptitude for organizational work seem to fit him admirably to assume this position in the AVMA at a time when developments in both veterinary medicine and agriculture are moving at an unprecedented rate.

Current Literature

ABSTRACTS

Glucose Metabolism in Calves

Some aspects of carbohydrate and electrolyte metabolism have been studied in 6 calves which were delivered from Holstein-Friesian cows with gestation periods of 35 to 62 days beyond normal.

The concentration of blood electrolytes was essentially normal, but the concentration of reducing sugars in the whole blood of the calves was abnormally low and decreased rapidly during the first 4 hours of extrauterine life. Since the decrease in blood fructose was essentially normal, a defective glucose metabolism is suggested. The deranged glucose metabolism was characterized by consistent hypoglycemia, even when the calves were fed at four-hour intervals or given intravenous infusions of glucose solutions. One calf also showed a lower glucose tolerance than did normal newborn calves.

All calves died or were killed in severe hypoglycemic states. When glucose was infused intravenously, the loss of glucose, potassium, and chloride in the urine was greater in these calves than in the normal newborn calf. The loss of glucose via the urine was not of sufficient magnitude to implicate renal mechanisms alone in the declining level of blood sugar found in the overdue calf. —[*Louis W. Holm: Some Aspects of Glucose Metabolism in Calves After Prolonged Gestation. Am. J. Vet. Res., 19, (Oct., 1958): 842-847.*]

Lungworm Infections in Calves

Subcutaneous injections of viable larvae produced lungworm (*Dictyocaulus viviparus*) disease in 5 experimental calves; the control calves remained negative. The larvae were injected in the cervical, flank, and gluteal regions. Signs of lungworm infection first appeared 13 to 17 days after the initial injection of larvae, with prepatent periods of 24 to 28 days. These infections were similar, clinically, to infections produced by oral administration of larvae to calves.—[*A. E. Wade and L. E. Swanson: Lungworm Infections in Calves Produced by Subcutaneous Injections of Larvae. Am. J. Vet. Res., 19, (Oct., 1958): 792-793.*]

Urolithiasis in Steers

The quantity of protein-bound hexosamine occurring in the urine of steers was measured by the hexosamine content of the precipitate formed in urine by phosphotungstic acid. Significant increases in the amounts of protein-bound hexosamine in the urine were shown to occur when the steers were shifted from an alfalfa hay ration to a fattening ration, when the concentrate to roughage ratio was increased from 4:1 to 8:1, and when the level of calcium in the ration was increased to three times the National Research Council requirements.

The two-way interactions, involving the high levels of concentrate to roughage with the calcium and calcium with phosphorus, produced significant increases in the urinary mucoproteins. The high level of phosphorus produced a highly significant acidification of the urine and significant reduction in the amounts of calculi recovered in the bladders, but had no effect upon the output of protein-bound hexosamine in the urine. An hypothesis is made that predisposition to calculosis can be measured by the level of mucoprotein in the urine. —[*R. H. Udall, A. W. Deem, and D. D. Maag: Studies on Urolithiasis. I. Experimental Production Associated with Feeding in Steers. Am. J. Vet. Res., 19, (Oct., 1958): 825-829.*]

Growth of Dwarf Beef Cattle

The administration of thyroactive iodinated protein, testosterone propionate, and diethylstilbestrol, singly or in combination, was carried out in attempts to stimulate growth in 16 osteochondrodystrophic-like dwarf Hereford cattle.

All of the hormone treatments appeared to produce a slight increase in rate of gain and to improve the general health of the dwarfs. In some instances, there was a slight increase in height at the withers. However, there was no indication that the dwarfism syndrome could be corrected by endocrine therapy with the thyroid, estrogenic, or androgenic hormones.—[*F. N. Andrews and J. M. Fransen: Effects of Endocrine Therapy on the Growth of Dwarf Beef Cattle. Am. J. Vet. Res., 19, (Oct., 1958): 822-824.*]

Survival of Infectious Organisms in Semen Extender

Survival of *Brucella abortus* strain 2308, *Leptospira pomona* strain 16-P54874, and *Vibrio fetus* strain K60453 inoculated into semen diluted with the Illini variable temperature (IVT) diluent and sealed in glass ampules was tested at 29 C.

Separate ampules inoculated with 100,000 organisms per milliliter were opened and cultured after 0, 48, and 96 hours of storage.

Control ampules contained IVT diluent without sulfanilamide and antibiotics. *Leptospira pomona* was viable only at the 0 time; *Br. abortus* and *V. fetus* were viable at 0, 48, and 96 hours of storage.

Viable *Br. abortus*, *L. pomona*, and *V. fetus* could not be demonstrated in the IVT diluent containing 0.003 Gm. of sulfanilamide, 500 I.U. of penicillin, and 500 μg. of dihydrostreptomycin sulfate per milliliter after 0, 48, and 96 hours of storage.—[*H. E. Rhoades and N. L. Van Demark: Survival of Brucella Abortus, Leptospira Pomona, and Vibrio Fetus in the Illini Variable Temperature Semen Extender at 29 C. Am. J. Vet. Res., 19, (Oct., 1958): 976-978.*]

Effect of Antibiotics on Chicken Spermatozoa

The effects of different levels of seven antibiotics on the motility of chicken spermatozoa, bacterial count, and fertility were studied.

Chloramphenicol, tetracycline, and oxytetracycline reduced motility at 450 and 900 units, or

micrograms, per milliliter, whereas penicillin, streptomycin, and oleandomycin did not reduce motility at levels as high as 900 units, or micrograms, per milliliter.

Results of plating semen samples, which had been treated with a single antibiotic and all possible two-way combinations, on eugon agar showed the greatest reduction in bacterial count when 450 units of penicillin was combined with 450 μg. of streptomycin or dihydrostreptomycin per milliliter, or when 90 μg. of tetracycline or oxytetracycline was combined with 90 μg. of streptomycin or dihydrostreptomycin per milliliter.

Similar, but less precise, results were obtained with thioglycollate broth.

The bacterial count of fresh chicken semen was found to be much higher than that reported for fresh bull semen.

There was a marked improvement in fertility of semen diluted with a buffer containing oxytetracycline and dihydrostreptomycin as compared with buffer without antibiotics and buffer with penicillin and dihydrostreptomycin.—[*F. H. Wilcox and Mary S. Shorb: The Effect of Antibiotics on Bacteria in Semen and on Motility and Fertilizing Ability of Chicken Spermatozoa. Am. J. Vet. Res., 19, (Oct., 1958): 945-949.*]

BOOKS AND REPORTS

The Closed Treatment of Common Fractures

This is the second edition, by a noted British orthopedic surgeon, of a book intended for resident casualty surgeons in human hospitals. The author re-emphasizes the nonoperative method and states that "far from being a crude and uncertain art, the manipulative treatment of fractures can be resolved into something of a science."

This may sound archly conservative, but it is not. The author feels that internal fixation or intramedullary nailing is mandatory in the treatment of fractures of the radius and ulna, lower third of the tibia, and midshaft and upper third of the femur. The point is that proper use of closed methods be made, and that the lure of being "up to date" should not cause the surgeon to use operative measures which may transgress the biological laws of the physiology of bone repair.

Much of the book deals with the preparation of plaster casts, and the specific treatment of certain common fractures of man; but there is a good review of the principles of bone healing which would be pertinent to the veterinary orthopedic surgeon.

The author decries the loss of skill at closed reduction and splinting which accompanies the increased interest in operative techniques. The situation in veterinary medicine, especially in the United States, is quite analogous.

This book is not for the veterinarian who wonders how that last case should have been treated, but it does broaden our understanding and gives us a perspective of our parent field. The book is well written, with good illustrations on fine paper. It is highly recommended as supplementary reading for veterinary orthopedic surgeons.—[*The Closed Treatment of Common Fractures. By John Charnley. 256 pages; illustrated. 2nd ed. Williams and Wilkins Co., Mt. Royal Ave., Baltimore, Md., 1957. Price $12.50.*]—R. L. LEIGHTON.

Zinsser's Bacteriology

The long shadow of a man is evident in the eleventh edition of Zinnser's bacteriology. This book has been the standard text in medical bacteriology since the first edition by Hiss and Zinsser in 1910. The present edition has the value of authentic contributions by eight authors.

Bacteriology has always been a rapidly developing science and has been given a tremendous boost with the advent of antibiotics. This new edition includes, under a new heading, the chemotherapy of each specific organisms as well as an up-to-date discussion of antibiotics. The favorable and unfavorable effects of ACTH and cortisone on each specific infection are included in this edition.

Zinsser's bacteriology has always been devoted to medical bacteriology and has not dealt to any significant extent with those bacteria pathogenic to the various species of animals unless they are also pathogenic to man. It is for this reason that this book may have limited value for the average veterinarian. However, those interested in immunology, bacterial physiology, and virology must include this text in their library.—[*Zinsser's Bacteriology. By David T. Smith, N. F. Conant et al. 953 pages. 11th ed. Appleton-Century-Crofts, Inc., New York, N.Y., 1957. Price $12.00.*]—I. A. MERCHANT.

Medical Radiation Biology

This work covers in a single volume the medical aspects of ionizing radiation and ultraviolet and visible light. It is designed for the general medical practitioner, the radiologist, the dentist, and the medical research worker. It would not have value for the veterinarian in general practice. However, any veterinarian engaged in medical research on either laboratory or domestic animals would find this book a worthwhile addition to his library. It emphasizes observations on radiation effects on man which are supplemented by experimental studies on other animals.

The book is divided into four sections: Part I, Fundamental Radiation Biology; Part II, Biology of Ionizing Radiation; Part III, Biology of Ultraviolet Radiation; and Part IV, Photobiology.

The work shows considerable attention to detail and is well organized. It is quite legible, the paper is good, and the type clear.

Two omissions detract somewhat from its usefulness and value. One is the absence of an index; secondly, there is missing in the section on ionizing radiation a discussion of RBE (relative biological effectiveness).—[*Medical Radiation Biology. By Friedrich Ellinger. 945 pages. Charles C Thomas, 301-327 E. Lawrence Ave., Springfield, Ill., 1957. Price $20.00.*]—W. C. BANKS.

THE NEWS

AVMA Professional Liability Insurance Again Available to Texas Members

After a lapse of ten years, the professional liability coverage which has been available without interruption to AVMA members in other states since 1925, can again be obtained by Association members in Texas. Advice to this effect was received recently at AVMA headquarters from Goerlich and Goerlich, the agents who originally developed the plan for the Association.

The insurance has not been available to Texas members since 1948 when the underwriters for the coverage withdrew operations in that state because of certain unacceptable changes in rules of the Texas Insurance Commission.

AVMA members in Texas who are interested may obtain a descriptive leaflet, application, and rate schedule by writing to the AVMA office, 600 S. Michigan Ave., Chicago 5, Ill.

Zoonoses Center Receives Rockefeller Foundation Support

The Pan American Sanitary Bureau announced recently that $10,000 has been made available by the Rockefeller Foundation to further the fight against zoonoses in the Americas. The money will be used to help finance the activities of a technical advisory group on zoonoses problems which are currently under study at the Pan American Zoonoses Center in Argentina.

The center was established in 1956 to help American nations reduce the burden to health and economy caused by zoonoses. It is the only institution of its kind and is located in Azul, Argentina, 185 miles south of Buenos Aires.

At present, its work is concentrated on rabies, brucellosis, and anthrax. The center receives financial and material support both from the Argentine government, as host country, and from international sources and is administered by the Pan American Sanitary Bureau.

The Rockefeller Foundation grant will enable the technical advisory group to develop recommendations for future control programs on zoonoses to be carried on by health and animal disease authorities of the Americas.

Dr. Benjamin D. Blood (COL '39) has been director of the Zoonoses Center since it began operations on Jan. 1, 1957.

Dr. C. M. Cooper Becomes New Editor of Veterinary Medicine

The board of directors of *Veterinary Medicine* have announced the selection of Dr. Carlos M. Cooper (ISC '49) as editor, effective October 1. He succeeds the late Dr. Robert L. Anderes, who suffered a heart attack on July 21, 1958, while attending a meeting in Chicago (*see* the JOURNALS, Aug. 15, 1958, p. 247, and Sept. 15, 1958, p. 341).

Dr. Cooper was a research director of Jensen-Salsbery Laboratories before joining *Veterinary Medicine*. He had become associated with Jensen-Salsbery in 1950 as assistant to Dr. Quin in professional service where he participated in virtually every phase of the company's operations with particular emphasis on product development and clinical medicine. Previously, Dr. Cooper had been engaged in practice in Montfort, Wis. He is a former editor of the *Iowa State College Veterinarian* and of the *Jensen-Salsbery Journal*.

Dr. Carlos M. Cooper

Veterinary Medicine, begun in 1905, was purchased by Dr. D. M. Campbell in 1910. In 1949, Dr. Anderes, Dr. Cooper's predecessor, acquired the magazine from the Campbell estate.

Mr. Charles Walters, Jr., publisher's assistant at *Veterinary Medicine*, said that it will now be Dr. Cooper's job to take it "on its next leg of the long-range trip" to further excellences.

General Ogle, Air Force Surgeon General, Retires

Major Gen. Dan C. Ogle, Air Force surgeon general for the past four years, retired on Nov. 30, 1958, after almost thirty years of active service.

A chief flight surgeon since 1932, General Ogle has dedicated himself to aeromedicine. Under his administration the Air Force has realized its goal for an Aeromedical Research

Center at Brooks Air Force Base, Texas, as well as the construction of 170 new worldwide medical facilities.

Born in Keithsburg, Ill., General Ogle attended local public schools. After receiving a baccalaureate degree in science and mathematics from Eureka College, Ill., in 1924, he returned to Keithsburg as an instructor and assistant principal of the high school. During this period he was elected to serve as mayor of Keithsburg.

After receiving his medical degree from the University of Illinois, College of Medicine, in 1929, he entered the U.S. Army Medical Corps as a first lieutenant. From 1929 through 1941, General Ogle served at William Beaumont General Hospital, El Paso, Texas; Chanute Field, Ill.; Luke Field, Hawaii; and Army and Navy General Hospital, Hot Springs, Ark.

In March, 1942, as Surgeon of the Technical Training Center at Miami Beach, Florida, General Ogle organized the service within the Miami area and developed a 3,000-bed hospital center. In December, 1944, he was assigned as surgeon of the Fifteenth Air Force in Italy where he pioneered the development of a medical service to support strategic Air Force operations. The following year he returned to the United States where he became surgeon at the Air University and instructor in medical subjects.

Upon graduating from the Air War College in June 1949, General Ogle was assigned as special assistant to the surgeon general and subsequently as deputy surgeon general. In March, 1953, General Ogle was named Surgeon of the United States Air Forces in Europe at Wiesbaden, Germany, and the following year was recalled to Washington to assume the position of Air Force Surgeon General.

In June of this year, General Ogle was awarded an honorary degree of Doctor of Laws by his alma mater, Eureka College. General Ogle's other awards include the Medailles d'Honneur du Service de Sante de l'Air from the French government and the Bronze Star.

General Ogle is a fellow of the American College of Physicians and the Aero Medical Association; and honorary fellow of the International College of Surgeons, the Southeastern Surgical Congress, the American College of Chest Physicians, and the American Association for the Surgery of Trauma; a diplomate of the American Board of Preventive Medicine with certification in Aviation Medicine; honorary director of the American Foundation for Tropical Medicine, Inc.; advisory vice-president of the Pan American Medical Association; a selector of the International Academy of Aviation Medicine, Inc.; a member of the National Board of Medical Examiners, the American Medical Association, the Association of Military Surgeons, the advisory board for Prevention of Asphyxial Death, the Gorgas Memorial Institute of Tropical and Preventive Medicine, the advisory editorial board for the *Journal of Aviation Medicine;* the Board of Regents for the National Library of Medicine; and the Alpha Omega Alpha; an honorary member of the Civil Aviation Medical Association, the Medical Society of the District of Columbia, the Washington Academy of Medicine, the Advisory Medical Board of the Leonard Wood Memorial, the American Venereal Disease Association, the American Veterinary Medical Association; and an advisory member of the Air Force Association Medical Advisory Council.

—U.S.A.F. Photo

Major Gen. Dan C. Ogle

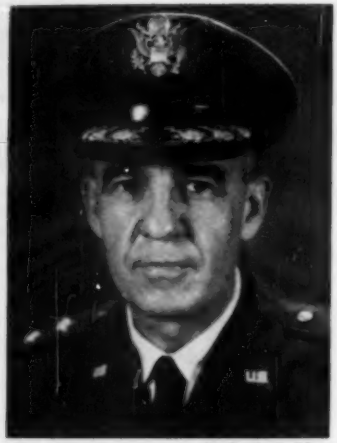
General Niess Named Air Force Surgeon General

Major Gen. Oliver K. Niess has been appointed surgeon general of the United States Air Force, effective Dec. 1, 1958.

Prior to his latest assignment, General Niess served as command surgeon of the Pacific Air Forces in Hawaii, a position he has held since September, 1954. During this tour of duty, General Niess had been instrumental in establishing medical care for the United States nationals throughout southeast Asia. He also organized and supervised the yearly Pacific Air Force Medical Conferences which are attended by representatives from the Asiatic countries.

Born in Belleville, Ill., in 1903, General Niess served his internship at the Fitzsimons General Hospital in Denver, Colo., and then was assigned to the Philippine department at Fort McKinley in the Philippine Islands.

General Niess is a graduate of the Army Medical Service School, the U.S.A.F. School of Aviation Medicine, and the Command and General Staff School. His current affiliations



—U. S. A. F. Photo
Major Gen. Oliver K. Niess

are: fellow of the American College of Surgeons; the American Medical Association; the Aero Medical Association; and the Association of Military Surgeons.

First Institute on Veterinary Public Health Practice

Some 200 veterinarians, physicians, sanitarians, and others in public health fields attended the First Institute on Veterinary Public Health Practice held in Ann Arbor, Mich., Oct. 6-9, 1958, under the auspices of the School of Public Health at the University of Michigan.

The four-day program was the outgrowth of two years of planning with the assistance of a number of agencies and groups including: American Public Health Association; Association of State and Territorial Health Officers; Association of State Public Health Veterinarians; American Board of Veterinary Public Health; American Veterinary Medical Association; Conference of Public Health Veterinarians; Department of the Air Force; Department of the Army; Michigan State University, College of Veterinary Medicine; Association of Military Surgeons; U. S. Public Health Service, Communicable Disease Center; Michigan Department of Health; Food and Drug Administration, H. E. W.; Conference of State and Territorial Epidemiologists; and Pan-American Sanitary Bureau, Regional Office for the Americas of WHO.

Beside the three-fold purpose of the Institute (*see* the JOURNAL, Sept. 15, 1958, p. 348), it was designed to serve all disciplines in public health, particularly state and local health department administrators, nurses, epidemiologists, nutritionists, health educators, environmental and occupational health, research, and laboratory personnel, together with veterinarians engaged in public health work.

In addition to the formal papers presented, registrants at the Institute were assigned to one of five section committees which held working sessions to develop information reflecting the conclusions of the Institute. These sections were:

I.—Working Relationship of Career Veterinarians to Other Disciplines in Public Health.

II.—Working Relationship of Veterinary Practitioners to Public Health.

III.—Actual and Potential Utilization of, and Contributions by, Career Public Health Veterinarians and Practicing Veterinarians in Official Public Health Programs.

IV.—Actual and Potential Utilization of, and Contributions by, Veterinarians in Voluntary and Official Agencies and Industry (other than Public Health).

V.—Education.

The program included more than 20 formal papers, some of which will be published in various professional journals, as well as in the proceedings of the Institute.

Among the program participants were the following: AVMA president R. E. Rebrassier; immediate past-president W. W. Armistead; former president W. O. Kester; former Executive Board chairman T. Lloyd Jones; and J. G. Hardenbergh, general consultant.

A New Serological Service for Dogs

A serological service which will permit the use of a nomograph in distemper vaccination and other tests to determine immunity to other diseases was announced by Dr. Thomas Benson, director of the diagnostic laboratory of the New York State Veterinary College at Cornell University. This service was made available on Oct. 15, 1958.

Serums from pregnant bitches are tested for antibody titers, and from the resulting nomograph constructed by the research staff of the Cornell Research Laboratory for Diseases of Dogs, the exact week in which expected litters can be successfully vaccinated against distemper will be determined.

Then one month after the vaccination, serums from two of the pups in the litter will be tested to see if immunity has been established. A nomograph, as defined by Webster, is a graph that enables one, by means of a straight edge, to read off the value of a dependent variable. In this case, the dependent variable is the earliest age to vaccinate pups against distemper and the value of the independent variable is the serum titer of the bitch.

Inasmuch as serum many times fails to provide adequate protection, the use of the nomograph to determine the exact week in which a

pup can be immunized becomes an important means of providing protection against distemper at the earliest possible age. This is expected to preclude the need for use of protective serum.

In addition to the nomograph test for distemper, other tests for immunity will be available for distemper, for infectious canine hepatitis, and for three types of leptospiras (*L. canicola, L. icterohaemorrhagiae,* and *L. pomona).*

As an aid to diagnosis, a serum sample may be procured during an acute illness and another sample about ten days later. An increase in serum titer in the second serum sample would furnish definite proof of infection with distemper, infectious canine hepatitis, leptospirosis, a combination of these, or something else.

This serological service is offered to veterinarians to enable them to diagnose disease more accurately, to increase their ability to protect dogs against distemper, and to determine the success of any vaccination procedure used in the prevention of distemper, infectious canine hepatitis, and leptospirosis. Details concerning fees and submission of serum specimens can be obtained from Dr. Benson at the N.Y. State Veterinary College, Cornell University.

s/HADLEY C. STEPHENSON, *Correspondent.*

Four State Groups Co-Sponsor Midwest Veterinarian-Nutrition Conference

The veterinary medical associations of Iowa, Kansas, Missouri, and Nebraska are joining with the Midwest Feed Manufacturers' Association of Kansas City to present the first area-wide nutrition conference planned especially for the veterinarian. This Midwest Veterinarian-Nutrition Conference will be held at the President Hotel in Kansas City, Mo., on Dec. 8-9, 1958. All veterinarians are invited to attend.

The following representatives from four veterinary medical associations are among those conferring in Kansas City, Mo., this December, to encourage and cement mutual understanding between the feed man and the veterinarian. Seated (left to right)—Drs. F. B. Young, Secretary of the Iowa V.M.A., Waukee, Iowa; Merle Henriksen, president of the Kansas V.M.A., Emporia, Kan.; and James H. Magilton, representing the Nebraska V.M.A., David City, Neb. Standing (left to right)—Drs. James K. Farrell, president of the Missouri V.M.A., Boonville, Mo.; and Chase Wilson (Ph.D.), chairman, Consumer's Cooperative Association in Kansas City.

Problems in the four areas of animal nutrition that are considered most important in today's practice will be covered by authorities in each field: swine, poultry, ruminants, and small animals. Dr. R. E. Rebrassier, president of the A.V.M.A., will speak at the banquet on the first evening of the conference sessions. The title of his presentation will be "The Relation of the Veterinarian to Industry".

Advance registrations may be sent in now to Midwest Feed Manufacturers' Association, 20 West 9th Street Building, Kansas City 5, Mo.

AMONG THE STATES AND PROVINCES

Arizona

State Association.—The regularly scheduled annual winter meeting of the Arizona V.M.A. will meet in Yuma, on Dec. 7-9, 1958. Veterinarians from the Rocky Mountain area and California are cordially invited to attend.

The roster of speakers planned for the meeting are: Drs. K. W. Smith, Colorado; L. Keith Wayt, Colorado; Richard L. Ott, Washington; Marvin J. Twiehaus, Kansas; C. H. Ozanian, California; Norman L. McBride, California; Leonard W. Dewhirst, parasitologist, Arizona; and Ned W. Rokey, Arizona.

s/ROBERT E. MCCOMB, JR., *Program Chairman.*

Arkansas

New Local Veterinary Medical Society Organized.—Eleven veterinarians of Little Rock, Ark., met at the Marion Hotel there, Sept. 16, 1958, and succeeded in organizing the Pulaski County Veterinary Medical Society. Previous efforts to form a veterinary society in Little Rock had not been successful.

The society drew up the following four objectives for its organization: the advancement of all phases of veterinary medicine within the state; establishment of a workable public relations program to be employed within the profession, with other professional organizations, and with the general public; maintenance of a close liason with the state and federal organizations for the control of animal diseases and with city, county, and state health departments; and the acquisition of support for a stronger state and national association.

Officers for the next twelve months are: Drs. William M. Taegel, Jr., Little Rock, president; Francis O. Garrett, Jr., North Little Rock, vice-president; and Harvie R. Ellis, 54 Belmont Drive, Little Rock, secretary-treasurer.

s/H. R. ELLIS, *Secretary-treasurer.*

California

Dean Jasper on Six-Month Leave.—Dr. Donald E. Jasper, dean of the School of Veterinary Medicine on the Davis campus of the University of California, has taken a six-month sabbatical leave.

In addition to working in the field of cell reaction to injury at the Armed Forces Institute of Pathology, Washington, D.C., Dean Jasper also plans to spend some time at the National Institutes of Health at Bethesda, Md.; Walter Reed Research Institute, Washington, D.C.; the U.S.D.A., ARS Laboratory at Beltsville, Md.; and the Plum Island Animal Disease Laboratory in New York.

Dr. Oscar W. Schalm, associate dean of the School of Veterinary Medicine, will be acting dean during Dr. Jasper's absence.—*The California Veterinarian, 12, (September-October, 1958).*

• • •

Dr. McFarland and Dr. Yeager Promoted as Dr. Boyd Retires.—Dr. Ray W. McFarland (WSC '39) has been appointed chief of the Bureau of Meat Inspection in the California Department of Agriculture, effective Oct. 1, 1958, to fill the vacancy created by the retirement of Dr. Geoffrey A. Boyd (COL '20) on September 12.

Dr. G. W. Yeager (TEX '48) has been appointed to the assistant chief's position vacated by the promotion of Dr. McFarland. Dr. Yeager's appointment was also effective October 1.

Dr. McFarland has been assistant chief of the bureau since February, 1956. Born in Chicago, Ill., he received his grade school, high school, and junior college education in Monrovia and Pasadena, Calif. Dr. McFarland has spent most of his professional career in meat inspection work, both in the federal service and in the state's department of agriculture.

After graduation, Dr. Yeager was engaged in private practice for one year. In June, 1949, he was appointed as a veterinarian in the Bureau of Livestock Disease Control, with headquarters in Turlock. He was transferred to the Bureau of Meat Inspection in September, 1951, and was assigned to supervise inspection work in a San Diego meat packing plant. Later, he was assigned relief duties and worked throughout Southern California.

A native of Los Angeles, Dr. Yeager received his earlier education in that city and completed his preveterinary work at the University of Oregon.

• • •

Alameda-Contra Costa Association.—The Alameda-Contra Costa V.M.A. met at Pland's Restaurant in Oakland, on Sept. 24, 1958, for a dinner meeting.

Dr. N. L. McBride of Pasadena presented a discussion on "Modern Surgical Techniques" and a film entitled "A Trip Through the Hospital."

s/GEORGE H. MULLER, *Secretary.*

• • •

Established Brucellosis Control Areas.—The following California Counties are now established brucellosis control areas: Alpine, Amador, Butte, Calaveras, Colusa, Del Norte, El Dorado, Glenn, Humboldt, Inyo, Lake, Lassen, Marin, Mendocino, Modoc, Mono, Monterey, Napa, Nevada, Placer, Plumas, Sacramento, San Benito, San Joaquin, Santa Clara, Santa Cruz, Shasta, Sierra, Siskiyou, Solano, Sonoma, Stanislaus, Sutter, Tehama, Trinity, Yolo, and Yuba.—*The California Veterinarian, 12, (September-October, 1958).*

Colorado

Denver Area Society.—The newly elected officers of the Denver Area Medical Society are as follows: Drs. Frank Judish, Denver, president; Vyrle Stauffer, Arvada, president-elect: Harvard E. Larson, Denver, vice-president; and Gene Bierhaus, Englewood, secretary-treasurer.

Dr. Richard Tolley, Englewood, was chosen to represent the society at the State Colorado V.M.A.

s/GENE M. BIERHAUS, *Secretary-Treasurer.*

District of Columbia

District Meeting.—The fourth annual meeting of the District of Columbia V.M.A. met Oct. 14, 1958, at the Sternberg Auditorium, Walter Reed Army Institute of Research, in Washington, D.C.

During the morning session at which Brig. Gen. Elmer W. Young, president, presided, the following speakers and their respective subjects were among those on the program: Dr. W. H. Eyestone, National Institutes of Health, Bethesda, Md.—Cancer Research in Domestic Animals; Dr. Kenneth McEntee, professor of obstetrics, Cornell University—Infertility in Cattle; and Donald E. Jasper, dean, College of Veterinary Medicine, University of California—Management of a Large Beagle Colony.

Dr. Erven A. Ross, committee on public relations, presided at the afternoon meeting of the Association. Subjects at this session included: various heat-lung pumps, plastic fixation of long bone fractures, and vascular graft. A buffet dinner in the evening capped the day's events.

Illinois

Captain Judy Retires from Dog World.—Captain Will Judy ended thirty-six years as editor of *Dog World* Magazine on Sept 10, 1958. The magazine was seven years old when the Judy Publishing Company purchased it; Captain Judy edited the first issue in January, 1923.

and with the last issue under his editorship, October, 1958, he embraced a total of 426 issues of the magazine.

He has also resigned as president and director of the Judy-Berner Publishing Company, which now owns all interest in *Dog World* (the Judy Publishing Company having sold its half interest) whose entire stock is now owned by George Berner, the new president and publisher of *Dog World*.

Captain Judy continues as president and director of the Judy Publishing Company, now located at 2517 S. Michigan Ave., Chicago, 16, after the disastrous fire on July 30, which destroyed almost the entire four-story Judy Building at 3323 Michigan Blvd., a mile south of the new location. Practically all contents and records were destroyed by the fire.

Continuing his interest in dog matters as president of National Dog Week, chairman of the Oldtimers of the Kennel World, and writer of a newspaper syndicated dog feature "Dog Comments," Captain Judy is currently engaged in getting the third edition of his work, *The Dog Encyclopedia*, to press.

• • •

Veterinary Symposium.—The director and staff of Gaines Dog Research Center sponsored the eighth veterinary symposium on "The Newer Knowledge About Dogs" held at the Kankakee Civic Auditorium in Kankakee on Oct. 22, 1958. Dr. C. A. Brandly, dean, College of Veterinary Medicine, University of Illinois, chairmanned the event.

Among the highlights on the program were: Drs. E. A. Majilton, Miami, Fla.—Early Surgery for Cataracts; C. H. Covault, Department of Veterinary Medicine and Surgery, Iowa State College—Effective Treatment for Strongyloidosis; Charles A. Slanetz, Institute of Comparative Medicine, Columbia University—Problems in Dog Diets; Robert W. Kirk, N. Y. State Veterinary College, Cornell University—The Therapeutics of Uremia; and W. V. Lumb, College of Veterinary Medicine, Michigan State University—Haemobartonellosis in Dogs.

At the end of the symposium, a tour of the Gaines Research Kennels was arranged for all interested parties.

Iowa

Central Iowa Association.—The second session of the Swine Disease Shortcourse sponsored by the Central Iowa V.M.A. at Iowa State College was held in the Memorial Union at I.S.C., Ames, on Oct. 20, 1958.

Swine erysipelas, swine brucellosis, leptospirosis, and hog cholera were among the subjects treated by the Iowa State's staff members during this second session.

s/S. L. HENDRICKS, *Secretary-Treasurer.*

Joint Association Meeting.—The twenty-first annual meeting of the Midwest Small Animal Association and region three meeting of the American Animal Hospital Association met at the Hotel Burlington Nov. 12-13, 1958, in Burlington.

In charge of the various sessions of the convention were: Drs. William G. Magrane, Mishawaka, Ind, president of the A.A.H.A.; Jack R. Dinsmore, Glenview, Ill., president of the Midwest Small Animal Association; and C. L. McGinnis, Peoria, Ill., program chairman. Dr. Wayne H. Riser, Skokie, Ill., moderated a floor discussion on the topic, "Veterinary Medicine in Changing Times" at the banquet held at the close of the two-day meeting.

A wide range of subjects included discussions on "Remodeling While Practice Continues" to "Digestive Disturbances."

Kansas

Kansas City Veterinary College's Meeting at Philadelphia.—Seventeen members of the K.C.V.C. alumni registered at the AVMA Convention in Philadelphia last August.

The 1917 and 1918 classes were best represented The secretary's and the treasurer's repor: were read during the meeting of the alumni's dinner-gathering and the matter of suspending publication of the *K.C.V.C. Alumni Quarterly* was referred to a committee composed of Drs. M. F. Wallace, Kansas City; H. J. Rollins, Raleigh, N. Car.; Ralph L. West, St. Paul, Minn.; and O. H. Person, Wahoo, Neb.

The officers for the past year were all reelected as follows: Drs. Clay C. Von Gremp, Decatur, Ga., president; George H. Leenerts, Humphrey, Neb., vice-president; and Charles D. Folse, Kemah, Texas, secretary-treasurer.—*K.C.V.C. Alumni Quarterly, 41, (September, 1958).*

• • •

Kansas City Association.—The regular monthly meeting of the Kansas City V.M.A. was held in the Aztec Room of the Hotel President, on Oct. 16, 1958, in conjunction with the Midwest Small-Animal meeting.

The program following the busiress session dealt with leptospirosis. The diagnosis, treatment, pathology, and prophylaxis of the disease were discussed by: Drs. Jacob E. Mosier and Marvin J. Twiehaus of Kansas State College; and Drs. James E. English and Kenneth H. Niemeyer of the University of Missouri.

Following the preliminary general discussion, the group was divided into large and small animal sections in order to cover group interests more effectively.

s/FRANK A. O'DONNELL, *Secretary.*

• • •

K. S. C. Enrollment Figures.—Kansas State College announced that for the first semester of the 1958-1959 college year, the enrollment in the four classes of the professional curriculum of

The Kansas Veterinary Medical Association displayed the above exhibit at the Kansas Free Fair in Topeka, Sept. 6-11, 1958, and the Kansas State Fair in Hutchinson, September 13-18.

Dr. M. W. Osburn, extension veterinarian and chairman of the Kansas V.M.A. committee on public relations, conducted the exhibit at both events, assisted by members of the association. Extension literature and 5,000 copies of the AVMA booklet, "Veterinary Medicine as a Career," were distributed at the two fairs.

• • •

the School of Veterinary Medicine was as follows: first year—70; second year—67; third year—70; fourth year—70.

In addition, there are also many students enrolled in the graduate school who are majoring in veterinary subjects.—*Veterinary Alumni News, 38, (Oct. 1, 1958).*

Missouri

Women's Auxiliary.—The Women's Auxiliary to the Missouri V.M.A. met for a luncheon and business meeting at the Hotel Tiger in Columbia, October 6.

The auxiliary's delegate to the AVMA National Convention in Philadelphia last August, Mrs. A. E. Blum of Crystal City, presented a concise report on the meeting and reported that the auxiliary was listed on the national honor roll this year.

A musical program introduced by the luncheon chairman, Mrs. Donald Rodabaugh, Columbia, included Mrs. Erma Wheelock, Mrs. H. H. Berrier, Mrs. C. J. Bierschwal, Jr., and Mrs. Paul Mathews.

Mrs. G. E. Bartholomees, Sheldon, introduced the honorary members, and special guests, who were presented with certificates of honorary membership in the auxiliary. They are: Mrs. D. C. Houser, Jasper; Mrs. J. L. Miller, Boonville; Mrs. H. C. Ware, Fulton; Mrs. J. C. Hann, Neosho; Mrs. G. S. Kirby, Warsaw; Mrs. Ashe Lockhart, Kansas City; Mrs. C. A. Schulz, Salem; Mrs. J. L. Wells, Blue Springs; Mrs. C. W. Strode, Ashe Grove; Mrs. Harry Hinds, Palmyra; Mrs. Ray Mier, Smithville; Mrs. O. S. Crisler, Columbia; Mrs. R. P. Poague, Shelbina; and Mrs. A. L. Abell, Monroe City.

s/MRS. W. H. MOWDER, *Correspondent.*

New York

Conference Activity at the Academy of Sciences.—The New York Academy of Sciences offered two conferences last November, 1958: one concerning "The Cytopathology of Virus Infection" was held November 7-8 and the other on "Hypothermia" on November 13-14. All sessions were conducted at the Barbizon-Plaza Hotel in New York.

Speakers at the conferences represented 19 states, Canada, England, and France. Informal subscription luncheons were held at the Academy Building during the course of both conferences.

• • •

New York City, Inc., Association.—The regular meeting of the Veterinary Medical Association of New York City, Inc., met Oct. 1, 1958, at the New York Academy of Sciences in New York City.

The entire program was devoted to discussion on pet birds. The following veterinarians with their respective subjects were included on the program: Drs. Irving Altman, practitioner, Brooklyn—Handling Pet Birds and Fracture Repair; Harry I. Goldwasser, practitioner, Woodside—Treatment Problems with Pet Birds; and Charles Gandal, Bronx Zoo Veterinarian, Yonkers—Demonstration and Presentation on Anesthesia of Pet Birds and the Surgical Removal of Subcutaneous Tumors.

Saskatchewan

Saskatchewan Association.—The annual convention of the Saskatchewan V.M.A. was held in Regina, Aug. 27-29, 1958, in commemoration of the fiftieth anniversary of the association.

Excellent papers and demonstrations were presented which included two papers on poultry conditions by Dr. C. H. Bigland of Edmonton, Alta., and two papers by Dr. Smith of the Ontario Veterinary College. Dominion and provincial affairs were discussed by Dr. Reeker of Ottawa and Dr. T. V. Johnson of Regina.

The committee on local arrangements consisted of Dr. Albelseth, chairman, and Drs. Carlson, Johnson, and Senior.—*The Veterinary News, 20, (September, 1958).*

South Carolina

Piedmont Association.—The Piedmont V.M.A. held regularly scheduled meetings during the months July, August, and September, 1958.

Presided over by Dr. Watson Matthews, Rock Hill, the following program speakers were presented at the meetings:

July.—Dr. F. P. Caughman, Jr., Columbia—Problems Confronting South Carolina Veterinarians.

August.—Dr. Edward Burns, State Hospital, Columbia—The Use of Modern Tranquilizing Agents in the Mental Patient.

September.—Hon. George Hart, Jr., City Councilman and banker, York—The Problems of Small Town Municipal Government.—*South Carolina Veterinarian, III, (October, 1958).*

• • •

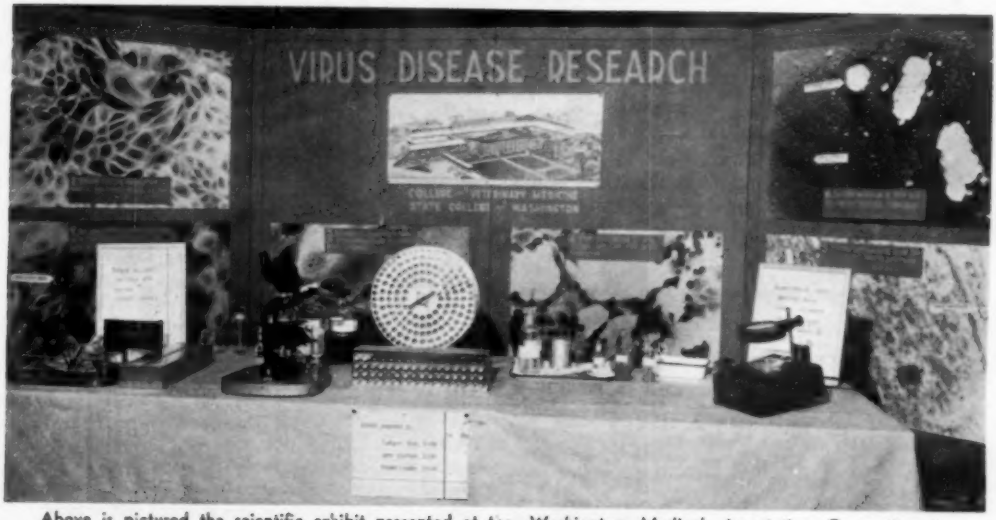
South Carolina Association of Veterinarians Preparing a Pet Booklet.—A committee headed by Dr. Herbert Riddle of Greenville is currently in the process of preparing a booklet geared for the use of pet owners.

It will be published in the name of the South Carolina Association of Veterinarians and will be made available to every practitioner in the state. The booklet will be particularly useful to the new pet owner. Consisting of an introductory section dealing with the problems of the newly arrived pup, a section on immunization, and a section on nutrition, this pamphlet will be published with the family pet in mind.—*South Carolina Veterinarian, III, (October, 1958).*

Virginia

Southwest Association.—The regular monthly meeting of the Southwest Virginia V.M.A. was held at the Animal Disease Laboratory in Blacksburg, on Oct. 2, 1958.

Washington



Above is pictured the scientific exhibit presented at the Washington Medical Association Convention in Spokane Sept. 15-17, 1958. It was prepared through the joint cooperation of Drs. Torbjorn Moll, John Gorham, and Robert Leader of the College of Veterinary Medicine at Washington State College.

The exhibit was well received by those attending the convention, and many felt that this active participation in the convention was a positive contribution to better public relations between the allied medical professions.

Dr. Cecil Howes, head of the poultry department at Virginia Polytechnic Institute, discussed "Vertical Integration in the Poultry Industry." The association plans a series of meetings in the future on vertical integration and its effect on the veterinary profession.

s/D. F. WATSON, *Secretary.*

Wisconsin

Joint Association Meeting.—A joint meeting of the Northeastern V.M.A. and the Wisconsin Valley V.M.A. was held at the Badger Breeders Cooperative Headquarters in Shawano, on Sept. 17, 1958. The Wisconsin Valley V.M.A. were the guests of the North East group. Approximately 100 veterinarians and their wives were present.

Drs. H. J. O'Connell, A. Erdmann, and W. J. O'Rourke addressed the meeting and Mr. Alton Dale Block, sire analyst for Badger Breeders Cooperative, reported on the working of his group in the breeding program.

The following officers were elected at the business meeting: Drs. B. A. Robinette, Coleman, president; W. E. Norris, Valders, vice-president; H. R. Trombley, Appleton, treasurer; W. Madson, Appleton, secretary; and K. W. Downey, Green Bay, trustee.

In the evening, the Badger Breeders Cooperative were hosts to the convention attendees at a dinner held at the Skalogoco Country Club. Mr. Kenneth Wallin, chairman of the State Board of Agriculture and general manager of the cooperative, spoke on present day dairy trends.

s/WILLIAM MADSON, *Secretary.*

U.S. GOVERNMENT

Veterinary Personnel Changes.—The following changes in the force of veterinarians in the U.S.D.A. are reported as of Sept. 26 and Oct. 3, 7, & 8, 1958.

TRANSFERS

John R. Burns, from Mason City, Iowa, to Menominee, Mich.
Donald L. Croghan, from St. Joseph, Mo., to Lansing, Mich.
William C. Davis, from Lansing, Mich., to Jackson, Miss.
Don Hinson Dixson, from Fort Worth, Texas, to Madison, Wis.
Robert A. Gale, from New York, N. Y., to Miami, Fla.
Dale C. Gigstad, from Topeka, Kan., to Omaha, Neb.
Carl D. Griffin, from Augusta, Ga., to St. Louis, Mo.
Jack E. Gross, from Fort Smith, Ark., to Greenville, S. Car.
John W. Howder, from Spokane, Wash., to Chicago, Ill.
William T. Hubbert, from Baltimore, Md., to St. Paul, Minn.
Willis H. Irvin, from Indianapolis, Ind., to New York, N. Y.
Ernest V. Maginnis, from St. Louis, Mo., to Fort Smith, Ark.
Dale D. Oshel, from Pearl River, N. Y., to Lansing, Mich.
Seidel N. Stephens, from Oklahoma City, Okla., to Sioux City, Iowa.
Albert W. Stichka, from Jacksonville, Fla., to San Francisco, Calif.
Charles J. Vosbrink, from Reno, Nev., to Madison, Wis.
Gilbert H. Wise, from Columbus, Ohio, to Sacramento, Calif.

RETIREMENTS

James W. Boylston, Columbia, S. Car.
Greydon S. Hicks, Fresno, Calif.

STATE BOARD EXAMINATIONS

Interested persons can obtain information about applications, fees, deadlines for filing applications, and exact time and place of examinations of the respective boards by writing to the persons whose names and addresses are given below.

TEXAS—Next licensing examinations will be held Jan. 14-16, 1959; Austin. The completed application must be received in the board office not later than 30 days before the examination date. Mr. T. D. Weaver, 207 Capital National Bank Building, Austin 16, executive secretary, State Board of Veterinary Medical Examiners.

DEATHS

Star indicates member of AVMA

★**Benjamin P. Chodos** (UP '01), 72, Harrisburg, Pa. (formerly of Lancaster, Pa.), a life member of the AVMA since 1957, died Sept. 9, 1958, in the Harrisburg Hospital, after a lengthy illness.

Born in Russia, Dr. Chodos was a pioneer in educating farmers to help prevent illness among their livestock. He began his career in Gap, later moving to service Lancaster County which he did for 45 years. He retired in February, 1956, and in April, moved to Harrisburg.

A member of the Pennsylvania State V.M.A., Christiana Lodge 47, F. & A.M., and the Masonic Order of Perfection, Dr. Chodos also belonged to the Conestoga Veterinary Club, the Lancaster Lodge of Elks, the University of Pennsylvania Alumni Club of Lancaster County, and the Conestoga Country Club.

Surviving Dr. Chodos are his widow, Estelle Astrich Chodos, a son, and a daughter.

David F. Coyner (OSU '11), 73, Fresno, Calif., a former city and county milk inspector, died Sept., 19, 1958.

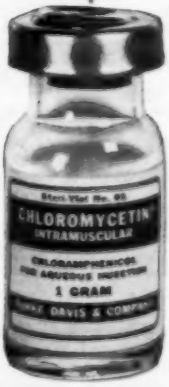
A native of Illinois, Dr. Coyner was named Fresno's meat inspector in 1921, becoming the city's milk inspector in 1925. Later, when the city and county milk inspection agencies were combined, he headed the department. He retired on June 1, 1956, after being honored at a dinner given by milk producers, milk inspectors, and fellow workers in the health department.

Surviving Dr. Coyner are his widow, Mrs. Clara Coyner, and two grandchildren.

CHLOROMYCETIN®
INTRAMUSCULAR

Chloramphenicol, PARKE-DAVIS

HIGH
CLINICAL EFFECTIVENESS with LOW BACTERIAL RESISTANCE



a specially prepared microcrystalline form of this **broad-spectrum** antibiotic, for suspension in an aqueous vehicle _ _ _

only by intramuscular injection

PARTICULARLY APPLICABLE TO VETERINARY MEDICINE

Professional literature will be sent on request

PARKE, DAVIS & COMPANY
DETROIT 32, MICHIGAN
TORONTO 14, ONTARIO

COMING MEETINGS

Animal Care Panel and the American Laboratory Animal Breeders Association. Annual meeting. Hamilton Hotel, Chicago, Dec. 3-4, 1958. Dr. N. R. Brewer, correspondent.

Nebraska State Veterinary Medical Association. Annual meeting. Hotel Cornhusker, Lincoln, Dec. 3-5, 1958. W. T. Spencer, 1259 North 37th, Lincoln, Neb., secretary-treasurer.

Arizona Veterinary Medical Association. Annual meeting. Yuma Country Club, Yuma, Dec. 7-9, 1958. Robert E. McComb, Jr., Phoenix, program chairman; Thomas E. Lightle, Route 1, Box 817, Yuma, in charge of reservations.

Kentucky Veterinary Medical Association. Winter meeting. Irvin S. Cobb Hotel, Paducah, Ky., Dec. 8-9, 1958. L. S. Shirrell, 545 E. Main St., Frankfort, Ky., secretary-treasurer.

American Association of Equine Practitioners. Annual meeting. LaSalle Hotel, Chicago, Ill., Dec. 15-16, 1958. M. L. Scott, 42 W. Market St., Akron 2, Ohio, secretary-treasurer.

Cornell University. Fifty-first annual conference for veterinarians. New York State Veterinary College, Ithaca, Jan. 7-9, 1958. W. A. Hagan, dean.

Southern California Veterinary Medical Association. Annual meeting. Beverly Hilton Hotel, Beverly Hills, Jan. 10, 1959. R. J. Schroeder, 1919 Wilshire Blvd., Los Angeles, Calif., program chairman.

Tennessee Veterinary Medical Association. Annual meeting. Noel Hotel, Nashville, Jan. 11-13, 1959. H. W. Hayes, 5009 Clinton Pike, Knoxville, secretary-treasurer.

Kansas Veterinary Medical Association. Fifty-fifth annual convention. Hotel Broadview, Wichita, Kan., Jan. 13-15, 1959. Dr. Maynard Curts, 5236 Delmar Ave., Kansas City 3, Kan., secretary.

Indiana Veterinary Medical Association. Annual meeting. Hotel Severin. Indianapolis, Ind., Jan. 14-16, 1959. George R. Burch, program chairman.

Iowa Veterinary Medical Association. Annual meeting. Hotel Fort Des Moines, Iowa, Jan. 20-22, 1959. F. B. Young, executive secretary.

Michigan State University. Thirty-sixth annual postgraduate conference for veterinarians. College of Veterinary Medicine, Michigan State University, East Lansing, Mich., Jan. 21-22, 1959. W. W. Armistead, dean.

Intermountain Veterinary Medical Association. Annual convention. Hotel Utah, Salt Lake City, Jan. 22-24, 1958. Douglas R. McKelvie, correspondent.

Oklahoma Veterinary Medical Association. Annual meeting. Mayo Hotel, Tulsa, Jan. 25-27, 1959. M. N. Riemenschneider, 122 State Capitol Bldg., Oklahoma City, secretary.

Minnesota State Veterinary Medical Society. Annual meeting. Leamington Hotel, Minneapolis, Jan. 26-28, 1959. B.S. Pomeroy, University of Minnesota, College of Veterinary Medicine, St. Paul 1, Minn. secretary-treasurer.

Louisiana State University. Annual conference. Pleasant Hill, Louniana State University Campus, Jan. 27-28, 1959. William T. Oglesby, correspondent.

Maryland State Veterinary Medical Association. Winter meeting. Emerson Hotel, Baltimore, Md., Jan. 27-28, 1959. Harry L. Schultz, Jr., secretary-treasurer.

California Veterinary Medical Association. Midwinter conference. School of Veterinary Medicine, University of California, Davis, Feb. 2-4, 1959. John W. Kendrick, conference chairman.

Ohio State Veterinary Medical Association. Annual convention. Neil House Hotel, Columbus, Feb. 4-6, 1959. Harry C. Sharp, 1411 W. Third Ave., Columbus, Ohio, executive secretary.

Illinois State Veterinary Medical Association. Annual meeting. LaSalle Hotel, Chicago, Feb. 16-18, 1959. C. B. Hostetler, 1385 Whitcomb Ave., Des Plaines, executive secretary.

Third Pan American Congress of Veterinary Medicine and Ninety-Sixth Annual Meeting, American Veterinary Medical Association. Joint meeting. Kansas City, Mo., Aug. 23-27, 1959. H. E. Kingman, Jr., executive-secretary, AVMA, 600 S. Michigan Ave., Chicago 5, Ill. B. D. Blood, secretary-general, Directing Council, Pan American Congress of Veterinary Medicine, P.O. Box 99, Azul, F.C.N.G.R., Argentina, S.A.

Foreign Meetings

International Veterinary Congress. Sixteenth session. Madrid, Spain, May 21-27, 1959. Prof. Pedro Carda A., general secretary, Calle Villanueva 11, Madrid.

U.S. COMMITTEE: Dr. W. A. Hagan, chairman, New York State Veterinary College, Ithaca, N. Y.; Dr. J. G. Hardenbergh, secretary, 600 S. Michigan Ave., Chicago 5, Ill.

Third World Congress on Fertility and Sterility Amsterdam, Holland, June 7-13, 1959. Dr. L. I. Swaab, Sint Agnietenstraat 4, Amsterdam, Holland, honorary secretary.

Regularly Scheduled Meetings

ALABAMA—Central Alabama Veterinary Association, the first Thursday of each month. Dr. G. W. Jones, Main St., Prattville, Ala., secretary-treasurer.

Jefferson County Veterinary Medical Association, the second Thursday of each month. S. A. Price, 213 N. 15th St., Birmingham, secretary.

APPLICATIONS

Applicants Not Members of Constituent Associations

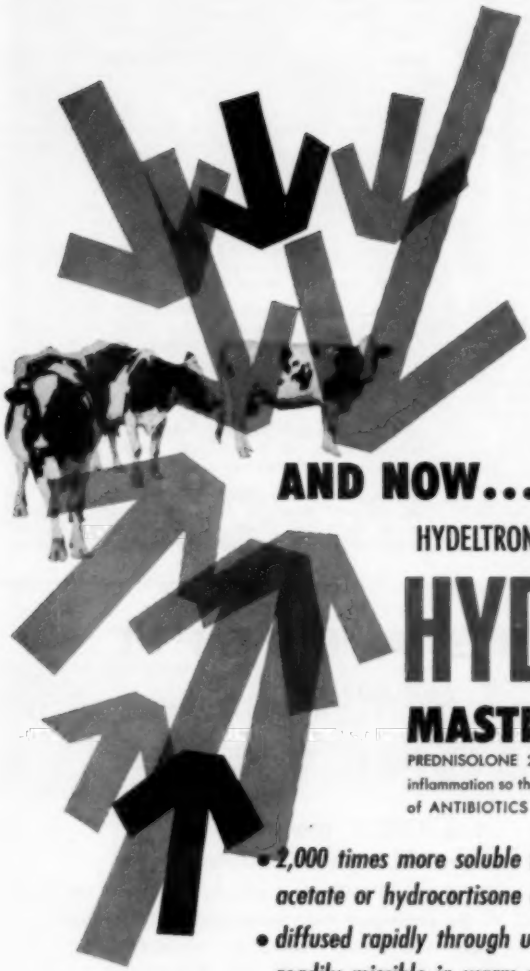
In accordance with paragraph (c) of Section 1, Article 1, of the Bylaws, the names of applicants who are not members of constituent associations shall be published in the JOURNAL. Written comments received by the Executive Secretary from any active member regarding the application as published, will be furnished to the Judicial Council for its consideration prior to acceptance of the application.

BLIZZARD, STEPHEN V. A.
199 Spadina Road, Toronto 5, Ont.
M.R.C.V.S., University of Edinburgh, 1953.
Vouchers: S. P. Bennett and P. E. Trainer.

FRIEDMAN, MARK H.
238 Clearmeadow Drive, East Meadow, L. I., N.Y.
D.V.M., Middlesex University, 1946.
Vouchers: R. J. Flynn and N. R. Brewer.

PEDE, ELMER R.
P. O. Box 65, Fort Belvoir, Va.
D.V.M., Michigan State University, 1942.
Vouchers: W. A. Limberger, Jr., and J. A. McCallam.

VALENZUELA, MANUEL R.
P. O. Box 9271, Mexico City 1, Mexico.
D.V.M., University of Mexico, 1947.
Vouchers: D. E. Davis and F. Cumago N.



AND NOW... *the unique benefits of the*

HYDELTRONE® *Family of Steroids in*

HYDELTRONE

MASTITIS OINTMENT with

PREDNISOLONE 21-PHOSPHATE® to suppress acute inflammation so that a BROAD-SPECTRUM combination of ANTIBIOTICS can get to the site of infection!

- *2,000 times more soluble than prednisolone acetate or hydrocortisone acetate*
- *diffused rapidly through udder tissue... readily miscible in warm milk*
- *aids in suppressing acute inflammation*

HYDELTRONE MASTITIS OINTMENT CHECKS INFLAMMATION
CONTROLS INFECTION—HELPS GET COWS BACK ON FULL MILK PRODUCTION—*FASTER!*

MERCK & CO., INC.
CHEMICAL DIVISION
RAHWAY, NEW JERSEY
VETERINARY

MERCK

Each 5 Gm. single-dose tube contains:

PROCAINE PENICILLIN	100,000 UNITS
DIHYDROSTREPTOMYCIN (AS THE SULFATE)	100 MG.
NEOMYCIN (AS THE SULFATE)	100 MG.
PREDNISOLONE 21-PHOSPHATE	5 MG.

SOLD TO VETERINARIANS ONLY

Mobile-Baldwin Veterinary Medical Association, the third Tuesday of each month. W. David Gross, 771 Holcombe Ave., Mobile, Ala., secretary.

North Alabama Veterinary Medical Association, the second Thursday of November, January, March, May, July, and September, in Decatur, Ala. Ray A. Ashwander, Decatur, Ala., secretary.

North East Alabama Veterinary Medical Association, the second Tuesday of every other month. Leonard J. Hill, P.O. Box 761, Gadsden, Ala., secretary-treasurer.

ARIZONA—Central Arizona Veterinary Medical Association, the second Tuesday of each month. Keith T. Maddy, Phoenix, Ariz., secretary.

Southern Arizona Veterinary Medical Association, the third Wednesday of each month at 7:30 p.m. E. T. Anderson, Rt. 2 Box 697, Tucson, Ariz., secretary.

ARKANSAS—Pulaski County Veterinary Medical Society, the second Tuesday of each month. Harvie R. Ellis, 54 Belmont Drive, Little Rock, Ark., secretary-treasurer.

CALIFORNIA—Alameda-Contra Costa Veterinary Medical Association, the fourth Wednesday of Jan., March, May, June, Aug., Oct., and Nov. Leo Goldston, 3793 Broadway, Oakland 11, Calif., secretary.

Bay Counties Veterinary Medical Association, the second Tuesday of February, April, July, September, and December. Herb Warren, 3004 16 St., San Francisco, Calif., executive secretary.

Central California Veterinary Medical Association, the fourth Tuesday of each month. R. B. Barsaleau, 2333 E. Mineral King, Visalia, Calif., secretary.

Kern County Veterinary Medical Association, the first Thursday evening of each month. James L. Frederickson, 17 Niles St., Bakersfield, Calif., secretary-treasurer.

Mid-Coast Veterinary Medical Association, the first Thursday of every even month. W. H. Rockey, P. O. Box 121, San Luis Obispo, Calif., secretary.

Monterey Bay Area Veterinary Medical Association, the third Wednesday of each month. Lewis J. Campbell, 90 Corral de Tierra, Salinas, Calif., secretary.

North San Joaquin Valley Veterinary Medical Association, the fourth Wednesday of each month at the Hotel Covell, in Modesto, Calif. Lyle A. Baker, Turlock, Calif., secretary.

Orange Belt Veterinary Medical Association, the second Monday of each month. Chester A. Maeda, 766 E. Highland Ave., San Bernardino, Calif., secretary.

Orange County Veterinary Medical Association, the third Thursday of each month. Donald E. Lind, 2645-N. Main St., Santa Ana, Calif., secretary.

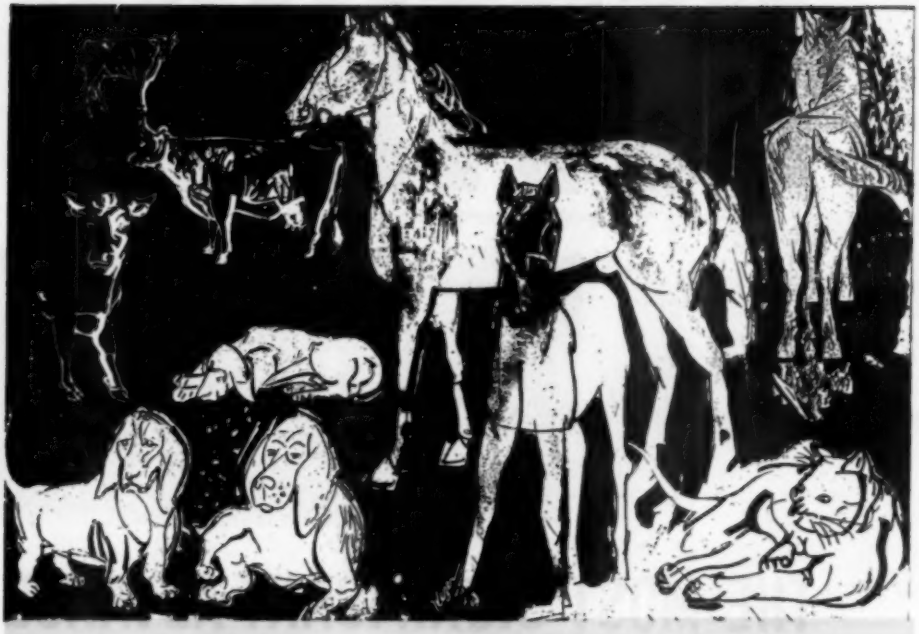
Peninsula Veterinary Medical Association, the third Monday of each month. R. M. Granfield, 2600 W. El Camino Real, San Mateo, Calif., secretary-treasurer.

Redwood Empire Veterinary Medical Association, the third Thursday of each month. Robert L. Chandler, P.O. Box 8, Ukiah, Calif., secretary.

Sacramento Valley Veterinary Medical Association, the second Wednesday of each month. W. E. Steinmetz, 4227 Freeport Blvd., Sacramento, Calif., secretary.

San Diego County Veterinary Medical Association, the fourth Tuesday of each month. H. R. Rossoll, 1795 Moore St., San Diego, Calif., secretary.

San Fernando Valley Chapter SCVMA, the second Tuesday of each month at 7:30 p.m., Hody's Restaurant, North Hollywood, Calif. Dr. V. H. Austin, 14931 Oxnard St., Van Nuys, secretary-treasurer.



prompt control of surface infections with

TOPICAL NITROFURANS

FURACIN®

brand of nitrofurazone

Prevention or treatment of bacterial infections of:

- surgical & traumatic wounds
- burns
- eye & ear
- cutaneous ulcers
- pyodermas
- osteomyelitis of compound fractures

FURACIN products contain FURACIN 0.2% in water-miscible vehicles. Available as FURACIN Dressing Veterinary in jars of 4 oz. and 1 lb.; FURACIN Dressing with Anesthetic (butacaine sulfate 0.5%) in tube of ½ oz. and jar of 1 lb.; FURACIN Soluble Powder in plastic "puff" bottle of 10 Gm.; FURACIN Solution in bottles of 2 oz.; 1 pint; 1 gal. FURACIN Ear Solution in bottles of 1 fl.oz.

FURASPOR®

brand of nitrofurfuryl methyl ether

Triple-action fungicide, sporicide, bactericide in:

- dermatomycoses and their secondary bacterial infections

Available as FURASPOR Liquid Veterinary, containing FURASPOR 0.4%, quaternary ammonium compound 0.5%, in a water-miscible vehicle of benzyl benzoate 15% and water. Odorless and nonstaining. Bottles of 2 fl.oz. with dauber top, and 1 pint.

NOTE: FURASPOR is *contraindicated* for cats and rabbits because of the benzyl benzoate content.

AVAILABLE THROUGH YOUR PROFESSIONAL VETERINARY DISTRIBUTOR

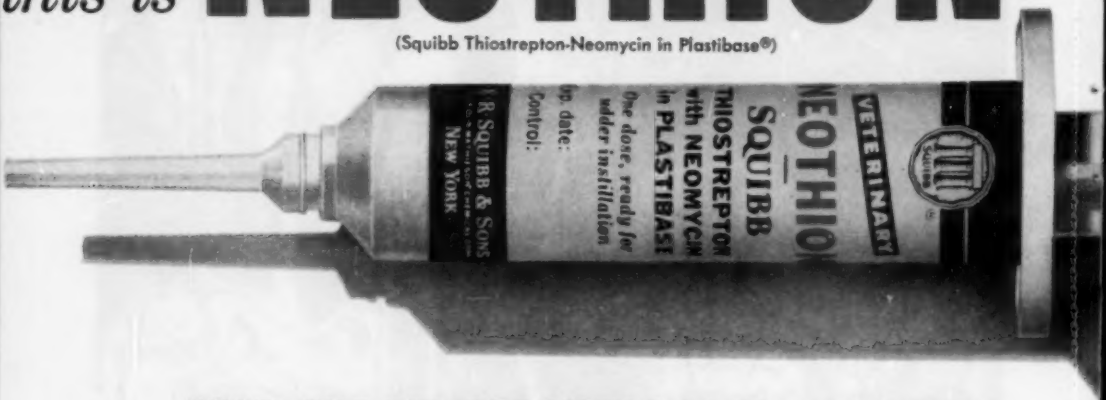
NITROFURANS—A NEW CLASS OF ANTIMICROBIALS—NEITHER ANTIBIOTICS NOR SULFONAMIDES

EATON LABORATORIES
NORWICH, NEW YORK

this is **NEOTHION** *

VETERINARY

(Squibb Thiostrepton-Neomycin in Plastibase®)



THE MOST SIGNIFICANT ADVANCE IN ANTI-MASTITIS THERAPY IN MANY YEARS

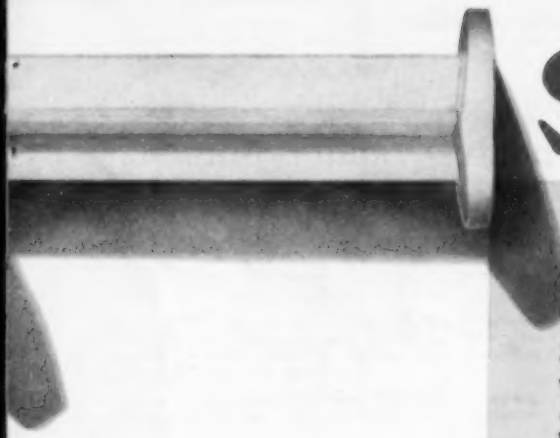
INTRODUCED LESS THAN A YEAR AGO, Neothion has already become one of today's thorough, dependable, *fast acting* formulations now available for anti-mastitis therapy. Neothion contains Squibb's new antibiotic, thiostrepton, plus neomycin, blended in a special non-irritating vehicle, Plastibase.

THE IMPORTANCE OF THIOSTREPTON. Thiostrepton now makes possible the control of mastitis caused by bacteria which have become resistant to other antibiotics. Thiostrepton is remarkably effective against gram-positive cocci.

Combined with neomycin, the resulting formula provides one of today's effective treatments for all types of bacterial mastitis, both chronic and acute. You can use Neothion with confidence. Neothion cannot harm the most delicate udder tissue and avoids the hazard of sensitization reactions to penicillin.

THE IMPORTANCE OF NEOMYCIN. Neomycin, of proved broad spectrum activity, provides the ideal adjuvant to thiostrepton. Together, these two antibiotics offer you a broader spectrum of antibacterial activity than any single antibiotic now available for mastitis therapy.

NEOTHION is available from your SQUIBB branch or your ethical wholesaler. NEOTHION IS SOLD ONLY TO VETERINARIANS, and is supplied in 1-dose syringes containing 50,000 units of thiostrepton plus 150 mg. of neomycin.



A NEW PRODUCT FROM

SQUIBB

NEOTHION

- **controls all types of bacterial mastitis**... both chronic and acute.
- **controls mastitis by penetrating the mastitis "resistance barrier"**... Neothion attacks bacteria which have become resistant to other antibiotics.
- **controls mastitis with lower dosage levels**... smaller amounts of the antibiotics in Neothion provide fast, more dependable therapy.
- **controls mastitis with a broader spectrum than any single anti-mastitis antibiotic now available**... broader than the triple sulfas, penicillin, streptomycin or neomycin.
- **controls mastitis safely**...Neothion cannot harm the most delicate udder tissue and avoids the hazard of sensitization reactions to penicillin.

NEOTHION IS THE ONLY ANTI-MASTITIS PRODUCT SOLD EXCLUSIVELY TO VETERINARIANS WHICH IS EFFECTIVE AGAINST ALL 9 TYPES OF MASTITIS-CAUSING BACTERIA

COMPARISON OF ACTIVITY OF SPECTRUM	GRAM-POSITIVE BACTERIA			GRAM-NEGATIVE BACTERIA					
	STAPHYLOCOCCI	PNEUMOCOCCI	STREPTOCOCCI	E. COLI	SHIGELLA	COLIFORMS	HEMOPHILUS	PROTEUS	PSEUDOMONAS
Triple Sulfas	▨	▨	▨	▨		▨	▨	▨	
Penicillin	▨	▨	▨						
Streptomycin			▨	▨	▨	▨	▨	▨	▨
Neomycin	▨		▨		▨	▨	▨	▨	▨
Brand A (NARROW SPECTRUM ANTIBIOTIC)	▨	▨	▨	▨	▨	▨	▨	▨	
Brand B (BROAD SPECTRUM ANTIBIOTIC)	▨	▨	▨	▨	▨	▨	▨	▨	
Brand C (BROAD SPECTRUM ANTIBIOTIC)	▨	▨	▨	▨	▨	▨	▨	▨	
NEOTHION	▨	▨	▨	▨	▨	▨	▨	▨	▨

THE IMPORTANCE OF PLASTIBASE. As a vehicle for thiostrepton and neomycin, Plastibase possesses unique physical properties and therapeutic advantages. Neothion is smooth and free-flowing. Neothion never freezes.

Unlike water-base products, Neothion does not mix with the milk, but floats *on* it and rises *with* it, thus bringing the full potency antibiotics in contact with infected upper udder tissues. As milk level drops, Neothion "clings" to udder tissues. Neothion does not milk out prematurely.

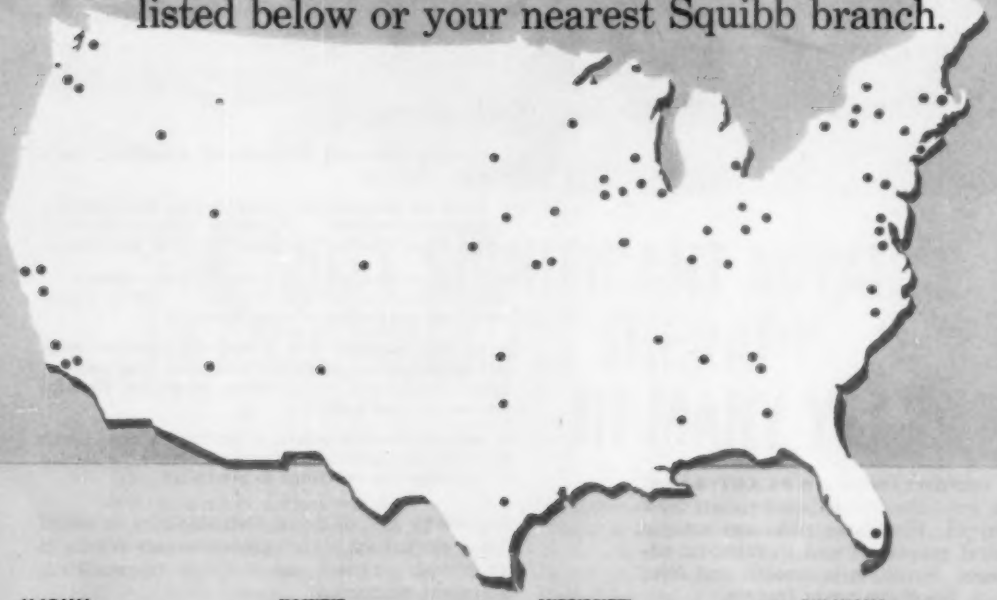
For additional information, write:
SQUIBB, *Veterinary Department,*
745 Fifth Avenue, New York 22, N. Y.

SQUIBB
—A NAME YOU CAN TRUST

YOU CAN GET NEOTHION FROM...
(turn page for name of your nearest veterinary supply house)

you can get NEOTHION

from any of the veterinary supply houses listed below or your nearest Squibb branch.



ALABAMA
Shoals Whse. Veterinary Supply Co., Florence

ARIZONA
Arizona Veterinary Supply Inc., Mesa

CALIFORNIA
H. C. Burns Co., Inc., Oakland
H. C. Burns Co., Inc, Los Angeles
California Medical Supply Co., Pasadena
California Veterinary Supply Co., Los Angeles
Central City Chemical Co. Consolidated, San Francisco
Sharpe & Vejar Co., Los Angeles
United Veterinary Corporation, Modesto
Valley Veterinary Supply Co., N. Hollywood

COLORADO
Rocky Mountain Veterinary Supply, Inc., Denver

DISTRICT OF COLUMBIA
District Veterinary Products Co., Washington

FLORIDA
Paul Dunt & Co., Inc., Miami Springs
L. A. Mosher, Inc., Miami

GEORGIA
Albany Serum Co., Albany
L. A. Mosher, Inc., Atlanta
L. A. Mosher, Inc., Albany
Southern Veterinary Supply Co., Rome

IDAHO
Snake River Valley Serum Co., Boise

ILLINOIS
Chicago Veterinary Supply, Inc., Chicago
Holmes Serum Co., [illegible]
Swingley Veterinary Supply Co., Freeport

INDIANA
Columbus Serum Co., Indianapolis
Missouri Valley Serum Co., Evansville

IOWA
Central Veterinary Supply Co., Des Moines
Missouri Valley Serum Co., Cedar Rapids

KANSAS
Missouri Valley Serum Co., Kansas City
Southwestern Serum Co., Wichita

KENTUCKY
L. A. Mosher, Inc., Louisville

LOUISIANA
Veterinary Supply Co., Inc., New Orleans

MARYLAND
A. J. Buck & Sons, Baltimore

MASSACHUSETTS
Jack Webster, Jr., Wakefield
David Yellen Co., Inc., Stoughton

MICHIGAN
Detroit Veterinary Supply Co., Detroit

MINNESOTA
Northland Veterinary Supply, St. Paul

MISSISSIPPI
Charles Horne Veterinary Supply Co., Newton

MISSOURI
Edwards Veterinary Supply Co., Kansas City
Ru-Vita Corporation, Kansas City

MONTANA
Mountain Veterinary Supply, Missoula

NEBRASKA
Blue Cross Serum Co., Superior
Grain Belt Supply Co., Omaha

NEW MEXICO
Great Western Serum, Albuquerque

NEW YORK
Gertrude DeVine Veterinary Supplies, Goshen
Essential Mineral Products Co., Batavia
Ethical Veterinary Supply Co., Long Island City
Farma-Ceuticals Company, East Homer
Lawler's Veterinary Supply Co., Carthage
Pennsylvania Veterinary Supply, Utica
Vetromart Co., Syracuse

NORTH CAROLINA
Barber Veterinary Supply, Fayette
L. A. Mosher, Inc., Raleigh

OHIO
Amco Drug Products, North Olmsted
W. A. Butler Company, Columbus
Columbus Serum Co., Inc., Columbus
P-T Veterinary Supply Co., Cincinnati
Zehr & Company, Pettisville

OKLAHOMA
Barber & Cochran Veterinary Supply Co., Oklahoma City

OREGON
H. C. Burns Co., Portland
Northwest Veterinary Supply Co., Oregon City
Portland Veterinary Supply Co., Portland

PENNSYLVANIA
Frank E. Lentz Co., Philadelphia
Pennsylvania Veterinary Supply, Harrisburg

SOUTH DAKOTA
Nelson Laboratories, Sioux Falls

TENNESSEE
L. A. Mosher, Inc., Memphis

TEXAS
Miller Veterinary Supply Co., Ft. Worth
So. Texas Veterinary Supply Co., San Antonio

UTAH
Mencimer Veterinary Supply Co., Ogden

VIRGINIA
Barber Veterinary Supply Co., Inc., Richmond

WASHINGTON
Cummings Medical Supply Co., Seattle
Northwest Veterinary Supply Co., Seattle

WISCONSIN
Wisconsin Biological Laboratories, Madison

TERRITORY OF HAWAII
Farm & Home Supply, Honolulu

San Fernando Valley Veterinary Medical Association, the second Friday of each month at the Casa Escobar Restaurant in Studio City. Dr. Rolf Reese, 23815 Ventura Blvd., Calabasas, Calif., secretary.

Santa Clara Valley Veterinary Association, the fourth Tuesday of each month. Kay Benlley, N. Fourth and Gish Rd., San Jose, Calif., secretary.

Southern California Veterinary Medical Association, the last Wednesday of each month. Don Mahan, 1919 Wilshire Blvd., Los Angeles 57, Calif., executive secretary.

Tulare County Veterinary Medical Association, the second Thursday of each month. D. E. Britten, 544 N. Ben Maddox, Visalia, Calif., secretary.

COLORADO—Denver Area Veterinary Medical Society, the fourth Tuesday of every month. Gene M. Bierhaus, 2896 S. Federal Blvd., Englewood, Colo., secretary-treasurer.

Northern Colorado Veterinary Medical Society, the first Wednesday of each month, in Fort Collins. Dr. James Voss, Veterinary Hospital, Colorado State University, Fort Collins, Colo., secretary.

DELAWARE—New Castle County Veterinary Association, the first Tuesday of each month at 9:00 p.m. in the Hotel Rodney, Wilmington, Del. A. P. Mayer, Jr., R.F.D. 2, Newark, Del., secretary-treasurer.

FLORIDA—Central Florida Veterinary Medical Association, the first Tuesday of each month, time and place specified monthly. Jack H. McFlyer, 5925 Edgewater Drive, Orlando, Fla., secretary.

Florida West Coast Veterinary Medical Association, the second Wednesday of each month at the Lighthouse Inn, St. Petersburg. William F. Casler, 2540 30th Ave., N., St. Petersburg, secretary-treasurer.

Jacksonville Veterinary Medical Association, the first Thursday of every month. Dodsons Restaurant, P. S. Roy, 4443 Atlantic Blvd., Jacksonville, Fla., secretary.

Northwest Florida Veterinary Medical Society, third Wednesday of each month, time and place specified monthly. T. R. Geci, 108B Catherine Ave., Pensacola, Fla., secretary.

Palm Beach Veterinary Society, the last Thursday of each month in the county office building at 810 Datura St., West Palm Beach. J. J. McCarthy, 500-25th Street, West Palm Beach, Fla., secretary.

Ridge Veterinary Medical Association, the fourth Thursday of each month in Bartow, Fla. Paul J. Myers, Winter Haven, Fla., secretary.

South Florida Veterinary Society, the third Wednesday of each month. Time and place specified monthly. Joe B. O'Quinn, 1690 E. 4th, Hialeah, Fla., secretary.

Suwannee Valley Veterinary Association, the fourth Tuesday of each month, Hotel Thomas, Gainesville. W. B. Martin, Jr., 3602 N. W. 6th St., Gainesville, Fla., secretary.

Volusia County Veterinary Medical Association, the fourth Thursday of each month. A. E. Hixon, 131 Mary St., Daytona Beach, Fla., secretary.

GEORGIA—Atlanta Veterinary Society, the third Thursday of each month at the Elk's Home, 726 Peachtree St., Atlanta. Donald C. Ford, Forest Park, secretary.

Georgia-Carolina Veterinary Medical Association, the second Monday of each month at 8:00 p.m., at the Town Tavern, Augusta, Ga. H. G. Blalock, Jr., 2190 Highland Ave., Augusta, secretary.

ILLINOIS—Chicago Veterinary Medical Association, the second Tuesday of each month. Charles H. Armstrong, 1021 Davis St., Evanston, secretary.

Eastern Illinois Veterinary Medical Association, the first Thursday of March, June, September, and December. A one-day clinic is held in May. Alfred G. Schiller, Veterinary Clinic, University of Illinois, Urbana, secretary-treasurer.

INDIANA—Central Indiana Veterinary Medical Association, the second Wednesday of each month. Peter Johnson, Jr., 4410 N. Keystone Ave., Indianapolis 5, secretary.

Michiana Veterinary Medical Association, the second Thursday of every month except July and December, at the Hotel LaSalle, South Bend, Ind. J. M. Carter, 3421 S. Main St., Elkhart, Ind., secretary.

Tenth District Veterinary Medical Association, the third Thursday of each month. J. S. Baker, P.O. Box 52, Pendleton, Ind., secretary.

IOWA—Cedar Valley Veterinary Medical Association, the second Monday of each month, except January, July, August, and October in Black's Tea Room, Waterloo, Iowa. A. J. Cotten, Grundy Center, secretary.

Central Iowa Veterinary Medical Association, the third Monday of each month, except June, July, and August, at 6:30 p.m., Breeze House, Ankeny, Iowa. John Herrick, Ames, secretary.

Coon Valley Veterinary Medical Association, the second Wednesday of each month, September through May, at 7:30 p.m., Cobblestone Inn, Storm Lake, Iowa. Robert McCutcheon, Holstein, secretary.

East Central Iowa Association, the second Thursday of each month at 6:30 p.m., usually in Cedar Rapids, Iowa. Dr. J. G. Irwin, Iowa City, secretary.

Fayette County Veterinary Medical Association, the third Thursday of each month at 6:30 p.m. in West Union, Iowa. H. J. Morgan, West Union, secretary.

Lakes Veterinary Association, the first Tuesday of each month, September through May, at 6:30 p.m., at the Gardson Hotel, Estherville, Iowa. Barry Barnes, Milford, secretary.

North Central Iowa Veterinary Medical Association, the third Thursday of April, at the Warden Hotel, Fort Dodge, Iowa. H. Engelbrecht, P. O. Box 797, Fort Dodge, secretary.

Northeast Iowa-Southern Minnesota Veterinary Association, the first Tuesday of February, May, August, and November at the Wisneslick Hotel, Decorah, Iowa, 6:30 p.m. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

Northwest Iowa Veterinary Medical Association, the second Tuesday of February, May, September, and December, at the Community Bldg., Sheldon. W. Ver Meer, Hull, secretary.

Southeastern Iowa Veterinary Association, the first Tuesday of each month at Mt. Pleasant, Iowa. Warren Kilpatrick, Mediapolis, secretary.

Southwestern Iowa Veterinary Medical Association, the first Tuesday of April and October, Hotel Chieftain, Council Bluffs, Iowa. J. P. Stream, Creston, secretary.

Upper Iowa Veterinary Medical Association, the third Tuesday of each month at 7:00 p.m., at All Vets Center, Clear Lake, Iowa. Richard Baum, Osage, secretary.

KENTUCKY—Central Kentucky Veterinary Medical Association, the first Wednesday of each month. R. H. Folsom, P.O. Box 323, Danville, Ky., secretary.

Jefferson County Veterinary Society of Kentucky, Inc., the first Wednesday of each month in Louisville or within a radius of 50 miles, except January, May, and July. G. R. Comfort, 2102 Reynolds Lane, Louisville, Ky., secretary-treasurer.

MARYLAND—Baltimore City Veterinary Medical Association, the second Thursday of each month, September through May (except December), at 9:00 p.m., at the

(Continued on adv. page 46)

Great-Granddaughter of Senator Vest at Dedication of "Old Drum" Statue

A crowd of 5,000 visitors and citizens of Warrensburg, Mo., jammed the Johnson County courthouse lawn on Sept. 27, 1958, the final day of National Dog Week, to witness the dedication of a statue of Old Drum, the hound dog made famous by the late Senator G. G. Vest's "Tribute to the Dog," at the Hornsby-Burden trial held in the Warrensburg courthouse 88 years ago.

Mrs. Converse Colt, Norwich, Vt., great-granddaughter of the late Senator G. G. Vest, at the dedication on the courthouse lawn in Warrensburg, Mo., of a statue of Old Drum, the hound dog made famous during an 1870 trial by the Senator's "Tribute to the Dog," perhaps the most popular piece of dog literature of all times. At left is Harry Miller, director of the Gaines Dog Research Center, New York.

Chief participant in the dedication was Mrs. Converse Colt of Norwich, Vt., a great-granddaughter of Senator Vest.

Mrs. Colt, flanked by Mr. Miller and Mayor H. H. Russell, of Warrensburg, rode in the lead car through the business section of town in a parade which preceded the dedication exercises. Captain Will Judy, of Chicago, made the presentation address, and John Swearingen, a drama student of Central Missouri State College, dressed in a costume of Senator Vest's era, recited the "Tribute to the Dog."

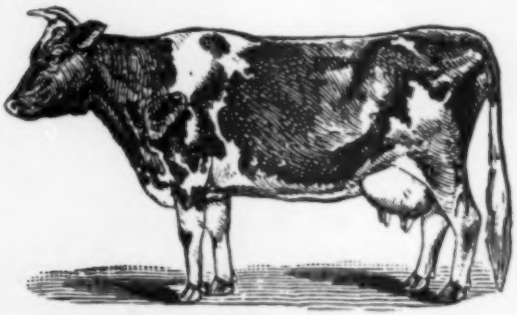
In the trial, Leonidas Hornsby was accused of shooting and killing a hound dog, Old Drum, belonging to his brother-in-law, Charles Burden. George Graham Vest, later a senator, so adroitly addressed the jury stressing a "dog's loyalty to his master is a virtue which can not be taken away with impunity," that the jury brought in a verdict in favor of the dog's owner, Mr. Burden, with damages beyond what he had claimed.

Senator Vest's speech is inscribed in full on the pedestal of the newly erected statue of Old Drum.

for wide-ranging control of many traumatic, eczematous and infectious skin conditions

Vioform®-Hydrocortisone CREAM

Broader control gives you better results: The comprehensive-complementary action of Vioform (fungicidal, bactericidal) plus hydrocortisone (anti-inflammatory, antipruritic) checks secondary pathogens while relieving exquisite pain and pruritus. *Because of low toxicity, you can give frequent applications to cats with ringworm.*



Nupercainal® OINTMENT

Used alone, or in combination with Vioform-Hydrocortisone Cream, Nupercainal Ointment relieves intolerable pain and pruritus (due to trauma, burns, infections) with soothing, long-lasting anesthetic action.

Dosage and Packaging: Refer to CIBA Veterinary Therapeutic Index.

Important: These drugs available only to graduate veterinarians through ethical distributors.

VIOFORM® (iodochlorhydroxyquin CIBA)
NUPERCAINAL® (dibucaine CIBA)

C I B A
SUMMIT, N. J.

FURADANTIN® VETERINARY

brand of nitrofurantoin

ORA-BOLS™

new, exclusive veterinary dosage form

- pleasing taste
  encourages prompt acceptance
- tapered shape permits easy swallowing
- readily retained

for small-animal urinary tract infections

FURADANTIN promptly produces high bactericidal concentrations in the urine; often there is marked improvement in a few days and complete recovery in 1-2 weeks.[1]

and canine tracheobronchitis

FURADANTIN administered for a period of 5 days effectively stopped coughing in 95% of 75 cases. In some instances symptoms were completely alleviated in 48 hours.[2] Recurrences have been very infrequent.

SUPPLIED: Bottle of 100 scored ORA-BOLS (50 mg. FURADANTIN with dextrose excipient). FURADANTIN VETERINARY also is available as: 10 mg. and 100 mg. bottles of 100 scored tablets and Oral Suspension containing 5 mg. per cc., bottle of 60 cc.

1. Belloff, G. B.: Calif. Vet. 9:27 (Sept.-Oct.) 1956. 2. Mosier, J. E.: Vet. Med. 52:445 (Sept.) 1957.

Available through your professional veterinary distributor.

NITROFURANS—a *new* class of antimicrobials—neither antibiotics nor sulfonamides

EATON LABORATORIES, NORWICH, NEW YORK

What Is Your Diagnosis?

Because of the interest in veterinary radiology, a case history and accompanying radiographs depicting a diagnostic problem are usually published in each issue.

Make your diagnosis from the picture below—then turn the page ▶

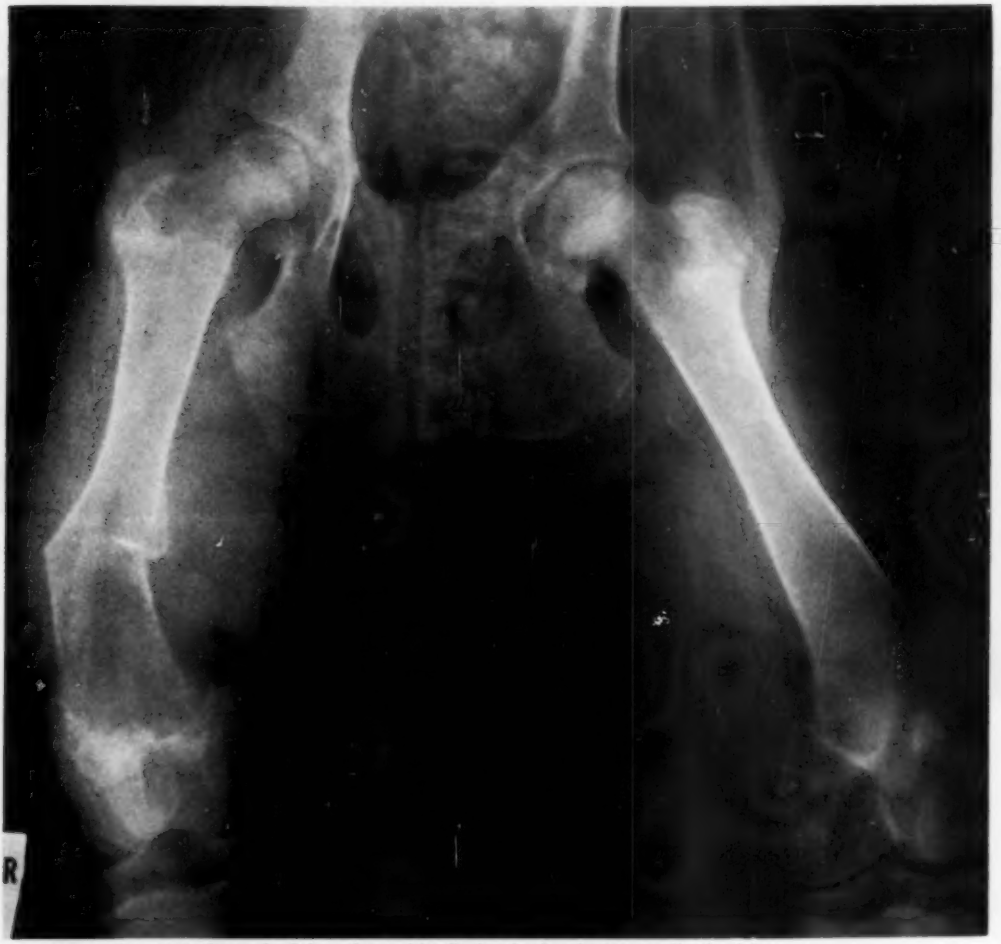


Fig. 1—Radiograph of the pelvis and hindlimbs of a female German Shepherd Dog.

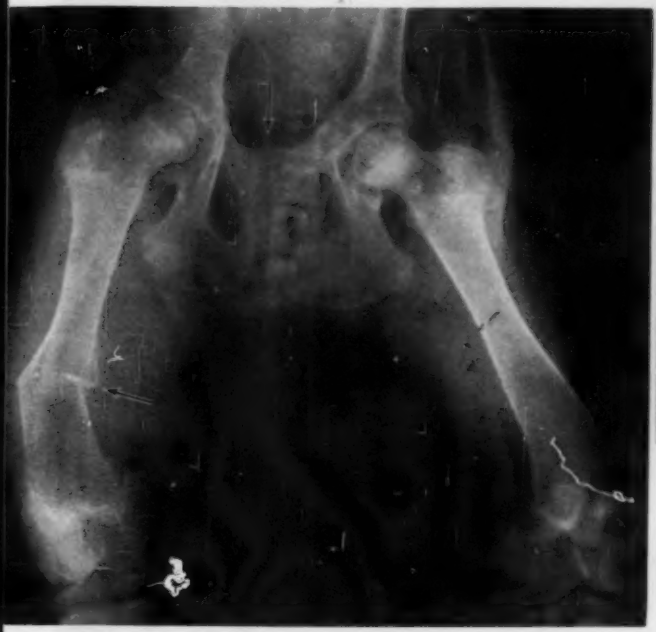
History.—A female German Shepherd Dog, 4 months old, developed normally in size and gait until 3 months of age. At that time it began to show increasing reluctance to play and to stand. The trouble seemed due to pain rather than weakness. The appetite, hair, and general appearance remained normal. Just previous to examination, the dog had fallen while walking and refused to rise. Radiographs of all four legs were taken.

Here Is the Diagnosis

(Continued from preceding page)

Diagnosis.—Idiopathic cortical hypoplasia of the bones, simulating osteogenesis imperfecta. Notice the fracture of the right hindleg and displacement of the pelvis..

Comment.—Osteogenesis imperfecta occurs in children and young small animals, particularly the dog and cat. In the pup and kitten, the condition occurs



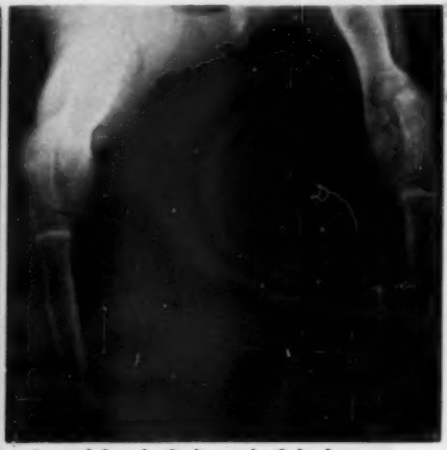


Fig. 2 (above)—Radiograph of the forelimbs of the dog showing cortical hypoplasia (osteogenesis imperfecta).

Fig. 3 (left)—Radiograph of the pelvis and hindlimbs showing cortical hypoplasia of the bones, which has resulted in a displaced pelvis and a "buckled type" fracture of the right femur (arrows).

between the eighth and twelfth week and begins with a slowed gait and lameness and soreness of the legs. Radiographs show retarded cortical development of all bones. Fractures are frequent, almost spontaneous, and often incomplete, as if the shaft had "buckled." The animal often continues to walk on the affected limb, which heals rapidly with little callus. Fractures of the spine also occur. There is no articular involvement. If the animal attains an age of 6 months, it begins to improve and the gait becomes normal. Fractures no longer occur and by the eighth or ninth month the cortices of the bones have a normal radiological appearance.

Laboratory analyses, on a limited number of affected dogs, showed the urine to be within normal limits, as were the blood calcium, phosphorus, and alkaline phosphatase. Some of the animals examined were slightly anemic and the total

(Continued on adv. page 46)

This case was presented by Dr. J. K. Bone, Chicago. Our readers are invited to submit histories, radiographs, and diagnoses of interesting cases which are suitable for publication.

(*Continued from adv. page 40*)

Park Plaza Hotel, Charles and Madison St., Baltimore, Md. Norman Herbert, 3506 Joann Drive, Baltimore 7, Md., secretary.

MICHIGAN—Central Michigan Veterinary Medical Association, the first Wednesday of every month at 7 p.m. Frank A. Carter, P.O. Box 78, Carson City, Mich., secretary.

Mid-State Veterinary Medical Association, the fourth Thursday of each month with the exception of November and December. Robert E. Kader, 5034 Armstrong Rd., Lansing 17, Mich., secretary.

Saginaw Valley Veterinary Medical Association, the last Wednesday of each month. S. Correll, Rt. 1, Midland, Mich., secretary.

Southeastern Veterinary Medical Association, the fourth Wednesday of every month, September through May. Gilbert Meyer, 14003 E. Seven Mile Rd., Detroit 5, Mich., secretary.

MISSOURI—Greater St. Louis Veterinary Medical Association, the first Friday of each month (except July and August), at the Coronado Hotel, Lindell Blvd. and Spring Ave., St. Louis, Mo., at 8 p.m. Edwin E. Epstein, 4877 Natural Bridge Ave., St. Louis 15, Mo., secretary.

Kansas City Veterinary Medical Association and Kansas City Small Animal Hospital Association, the third Thursday of each month at the Hotel President, Kansas City, Mo. Frank A. O'Donnell, Parksville, Mo., secretary-treasurer.

NEVADA—Western Nevada Veterinary Society, the first Tuesday of each month. Paul S. Silva, 1170 Airport Road, Reno, Nev., secretary.

NEW JERSEY—Central New Jersey Veterinary Medical Association, the second Thursday of November, January, March, and May at Old Hights Inn, Hightstown, N. J. David C. Tudor, Cranbury, N. J., secretary.

Metropolitan New Jersey Veterinary Medical Association, the third Wednesday evening of each month from Octo-

(*Continued from adv. p. 44*)

blood proteins were slightly decreased.[1] These findings, however, are consistent with the averages seen in pups that are considered normal.

In a group of 28 kittens subjected to treatment, all thrived except 2 that died of enteritis. The treatment consisted of confining them to a small area, providing a good diet and proper sex hormones.[2]

This condition is thought to be hereditary but the exact cause is not known. Animals from families that produce this defect should not be used for breeding.

References

[1]Calkins, E., Kahn, D., and Diner, W. C.: Idiopathic Familial Osteoporosis in Dogs: "Osteogenesis Imperfecta." Ann. New York Acad. Sci., *64*, (Aug. 17, 1956): 410-423.

[2]Coop, M. C.: A Treatment for Osteogenesis Imperfecta in Kittens. J.A.V.M.A., *132*, (April 1, 1958): 299-300.

AVMA Research Fellowships Available

The Council on Research of the AVMA announces the availability of a number of fellowships for postgraduate training for the academic year, 1959-1960.

The recipient of a fellowship must be a veterinarian and a citizen of the United States or Canada. Veterinary students who expect to graduate at the end of the current school year and who wish to follow a career in research may apply for a fellowship.

The latest date for filing the completed application is Feb. 15, 1959. Approximately one month is required for processing completed applications after receipt by the secretary of the Council. Qualified persons should secure and submit applications as early as possible to insure their file being complete for presentation to the Committee on Fellowships.

The Committee on Fellowships of the Council on Research will meet in March to consider applications, and the awards will be announced soon afterward. The stipend will be determined in each case by the needs of the individual, the location of the school in which he proposes to work, and other factors. In general, the stipends range from $100 monthly and upward.

Any qualified person interested in graduate training may obtain application blanks and other information by writing to Secretary, AVMA Council on Research, C. H. Cunningham, College of Veterinary Medicine, Michigan State University, East Lansing, Mich.

ber through April, except December, at the Irvington House, 925 Springfield Ave., Irvington, N.J. Bernard M. Weiner, 787 Clinton Ave., Newark, N.J., secretary.

Northern New Jersey Veterinary Association, the fourth Tuesday of each month at the Elks Club, Hackensack. Burritt Lupton, 569 Franklin Ave., Wyckoff, secretary.

Northwest Jersey Veterinary Society, the third Wednesday of every odd month. G. L. Smith, P.O. Box 938, Trenton, N.J., secretary.

Southern New Jersey Veterinary Medical Association, the fourth Tuesday of each month at the Collingswood Veterinary Hospital, Collingswood. R. M. Sauer, secretary.

Pfizer Laboratories Appoints New Veterinary Medical Director

Appointment of Dr. Hilmer L. Jones as veterinary medical director of Pfizer Laboratories was announced Oct. 4, 1958, by Dr. J. J. Van Gasse, general manager of the pharmaceutical division of Charles Pfizer & Co., Inc.

Dr. Hilmer L. Jones

Dr. Jones, who joins the company's headquarters staff in Brooklyn, N.Y., will coordinate clinical investigation of new Pfizer veterinary products and will serve as technical consultant to veterinarians in private practice.

Formerly a research veterinarian at Pfizer's agricultural research and development center near Terre Haute, Ind., Dr. Jones holds B.S., M.S., and D.V.M. degrees from Alabama Polytechnic Institute.

While at Terre Haute, Dr. Jones worked in agricultural research and as a field veterinarian, testing the practical application of new research discoveries. During this time he practiced under a variety of conditions encountered by poultry specialists as well as by large and small animal veterinarians.

In his new position, Dr. Jones will also serve as liaison with Pfizer scientists at Terre Haute and will help guide veterinary products from research to marketed dosage form.

NEW MEXICO—Bernalillo County Veterinary Practitioners Association, third Wednesday of each month, Fez Club, Albuquerque, N.M. Jack Ambrose, 3018 N. Rio Grande Blvd., Albuquerque, secretary-treasurer.

NEW YORK—New York City, Inc., Veterinary Medical Association of, the first Wednesday of each month at the New York Academy of Sciences, 2 East 63rd St., New York City. C. E. DeCamp, 43 West 61st St., New York 23, N. Y., secretary.

New York State Veterinary College, Annual conference for veterinarians. Cornell University, Ithaca. W. A. Hagan, New York State Veterinary College, Cornell University, Ithaca, N. Y., dean.

Monroe County Veterinary Medical Association, the first Thursday of even-numbered months except August. Irwin Bircher, 50 University Ave., Rochester, N. Y., secretary.

NORTH CAROLINA—Central Carolina Veterinary Medical Association, the second Wednesday of each month at 7:00 p.m. in the O'Henry Hotel, Greensboro. W. R. Dobbs, Box 869, Albemarle, secretary.

Eastern North Carolina Vererinary Medical Association, the first Friday of each month, time and place specified monthly, Byron H. Brow, Box 453, Goldsboro, N. Car., secretary.

Piedmont Veterinary Medical Association, the last Friday of each month. T. L. James, Box 243, Newton, N. Car., secretary.

Twin Carolinas Veterinary Medical Association, the third Friday of each month at Orange Bowl Restaurant, Rockingham, N. Car., at 7:30 p.m. J. E. Currie, 690 N. Leak St., Southern Pines, N. Car., secretary.

Western North Carolina Veterinary Medical Association, the second Thursday of every month at 7:00 p.m. in the George Vanderbilt Hotel, Asheville, N. Car. Vilu Lind, 346 State St., Marion, N. Car., secretary.

OHIO—Cincinnati Veterinary Medical Association, the third Tuesday of every month at Shuller's Wigwam, 6210 Hamilton Ave., at North Bend Road, G. C. Lewis, Cincinnati, Ohio, secretary-treasurer.

AMERICAN VETERINARIANS FOR ISRAEL
announces
Special Charter Flight, May 10-June 5, 1959
ISRAEL, SPAIN, EUROPE

ISRAEL 10th Anniversary Celebration Veterinary Symposium and dedication of new Veterinary Institute; tours.

SPAIN International Veterinary Congress in Madrid; tours.

EUROPE Post-congress tours to Paris and London.

Special Excursion Air Rates Number limited
For further particulars write
Dr. H. C. Newman, Box 145, Merrifield, Va.

Columbus Academy of Veterinary Medicine, every month, September through May. E. M. Simonson, Columbus, Ohio, secretary-treasurer.

Cuyahoga County Veterinary Medical Association, the first Wednesday in September, October, December, February, March, April and May, at 9:00 p.m. at the Carter Hotel, Cleveland, Ohio. F. A. Coy, Cleveland, Ohio, secretary.

Dayton Veterinary Medical Association, the third Tuesday of every month. O. W. Fallang, Dayton, secretary.

Killbuck Valley Veterinary Medical Association, the first Wednesday of alternate months beginning with February. D. J. Kern, Killbuck, Ohio, secretary-treasurer.

Mahoning County Veterinary Medical Association, the fourth Tuesday of each month, at 9:00 p.m., Youngstown Maennerchor Club, Youngstown, Ohio. Sam Segall, 2935 Glenwood Ave., Youngstown, secretary.

Miami Valley Veterinary Medical Association, the first Wednesday of December, March, June, and September. J. M. Westfall, Greenville, Ohio, secretary-treasurer.

North Central Ohio Veterinary Medical Association, the last Wednesday of each month except during the summer. R. W. McClung, Tiffin, Ohio, secretary-treasurer.

Northwestern Ohio Veterinary Medical Association, the last Wednesday of March and July. C. S. Alvanos, Toledo, Ohio, secretary-treasurer.

Stark County Veterinary Medical Association, the second Tuesday of every month, at McBrides Emerald Lounge, Canton, Ohio. M. L. Willen, 4423 Tuscarawas St., Canton, Ohio, secretary.

Summit County Veterinary Medical Association, the last Tuesday of every month (except June, July, and August), at the Mayflower Hotel, Akron, Ohio. M. L. Scott, Akron, Ohio, secretary-treasurer.

Tri-County Veterinary Medical Association, the fourth Wednesday of January, May, and September. Mrs. R. Slusher, Mason, Ohio, secretary-treasurer.

OKLAHOMA—Oklahoma County Veterinary Medical Association, the second Wednesday of every month, 7:30 p.m., Patrick's Foods Cafe, 1016 N.W. 23rd St., Oklahoma City. Forest H. Stockton, 2716 S.W. 29th St. Oklahoma City, Okla., secretary.

Tulsa Veterinary Medical Association, the third Thursday of each month in Directors' Parlor of the Brookside State Bank, Tulsa, Okla. Arlen D. Hill, 5302 E. 11th St., Tulsa, Okla., secretary.

Tulsa Association of Small Animal Veterinarians, first and third Mondays, City-County Health Dept. T. E. Messler, 3104 E. 51st St., Tulsa, Okla., secretary.

The dangerous, disconcerting interruption in surgery can make the difference between success and failure for the veterinarian—life or death to the patient. And it's an extremely rare occurrence with a reliable, safe anesthetic like

short-acting

NEMBUTAL®

(Pentobarbital, Abbott)

Practicing veterinarians over a quarter century have learned to depend on the uniformity and purity of this short-acting anesthetic. When you use it, Doctor, you'll find—

Onset of action is prompt—surgical hypnosis being achieved in just 3 to 5 minutes from a single intravenous dose.

Degree of cerebral depression and duration of anesthesia are easily controlled, lasting from 45 to 90 minutes.

Optimal skeletal muscle relaxation accompanying NEMBUTAL anesthesia facilitates surgery; interruptions during procedures are rare.

Side effects are minimal with safe, easy-to-use NEMBUTAL; and there is little danger of cumulative effect with this short-acting agent.

Order safe, reliable, uniform NEMBUTAL from your Abbott representative, your distributor or from Veterinary Division, Abbott Laboratories, North Chicago, Illinois.

Abbott

OREGON—Portland Veterinary Medical Association, the second Tuesday of each month, at 7:30 p.m. Ireland's Restaurant, Lloyds', 718 N.E. 12th Ave. Portland. Donald L. Moyer, 8415 S.E. McLoughlin Blvd., Portland 2, Ore., secretary.

Williamette Veterinary Medical Association, the third Tuesday of each month, except July and August, at the Marion Hotel, Salem. Marvin M. Corff, McMinnville, Ore., secretary.

PENNSYLVANIA—Keystone Veterinary Medical Association, the fourth Wednesday of each month at the University of Pennsylvania School of Veterinary Medicine. Raymond C. Snyder, N.E. Corner 47th St. and Hazel Ave., Philadelphia 43, Pa., secretary.

Lehigh Valley Veterinary Medical Association, the first Thursday of each month. Stewart Rockwell, 10th and Chestnut Sts., Emmaus, Pa., secretary.

Pennsylvania Northern Tier Veterinary Medical Association, the third Wednesday of each odd numbered month. R. L. Michel, Troy, Pa., secretary.

SOUTH CAROLINA—Piedmont Veterinary Medical Association, the third Wednesday of each month at the Fairforest Hotel, Union, S. Car. Worth Lanier, York, S. Car., secretary.

Georgia-Carolina Veterinary Medical Association—see GEORGIA.

TEXAS—Coastal Bend Veterinary Association, the second Wednesday of each month. J. Marvin Prewitt, 4141 Lexington Blvd., Corpus Christi, Texas, secretary.

VIRGINIA—Central Virginia Veterinarians' Association, the third Thursday of each month at the William Byrd Hotel in Richmond at 8:00 p.m. M. R. Levy, 312 W. Cary Ct., Richmond 20, Va., secretary.

Northern Virginia Veterinary Conference, the second Tuesday of each month. Francis E. Mullen, 1130 S. Main St., Harrisonburg, Va., secretary-treasurer.

Northern Virginia Veterinary Society, the second Wednesday of every third month. Meeting place announced by letter. H. C. Newman, Box 145, Merrifield, secretary.

Southwest Virginia Veterinary Medical Association, the first Thursday of each month. D. F. Watson, Blacksburg, secretary.

WASHINGTON—Seattle Veterinary Medical Association, the third Monday of each month, Magnolia American Legion Hall, 2870 32nd W., Seattle. Roy C. Toole, 10415 Main St., Bellevue, secretary.

South Puget Sound Veterinary Association, the second Thursday of each month except July and August. B. D. Benedictson, 3712 Plummer St., Olympia, Wash., secretary.

WEST VIRGINIA—Kyowva (Ky., Ohio, W. Va.) Veterinary Medical Association, the third Thursday of each month in the Hotel Pritchard, Huntington, W. Va., at 8:30 p.m. Harry J. Fallon, 200 5th St., W. Huntington, W. Va., secretary.

WISCONSIN—Central Wisconsin Veterinary Medical Association, the second Tuesday of each quarter (March, June, Sept., Dec.). D. F. Ludvigson, Ridgeland, Wis., secretary.

Dane County Veterinary Medical Association, the second Thursday of each month. Dr. E. P. Pope, 409 Farley Ave., Madison, Wis., secretary.

Milwaukee Veterinary Medical Association, the third Tuesday of each month, at the Half-Way House, Blue Mound Rd. Dr. R. H. Steinkraus, 7701 N. 59th St., Milwaukee, Wis., secretary.

Northeastern Wisconsin Veterinary Medical Association, the third Wednesday in April. William Madson, 218 E. Washington St., Appleton, Wis., secretary.

Rock Valley Veterinary Medical Association, the first Wednesday of each month. W. E. Lyle, P. O. Box 107, Deerfield, Wis., secretary.

Southeastern Veterinary Medical Association, the third Thursday of each month. John R. Curtis, 419 Cook St., Portage, Wis., secretary.

Wisconsin Valley Veterinary Medical Association, the second Tuesday of every other month. E. S. Scobell, Rt. 2, Wausau, Wis., secretary.

Schering Research Grants Total $431,000

Grants for independent, scientific, and medical research totaling $431,000 were made by Schering Corporation, pharmaceutical manufacturer, during the first six months of 1958, according to a recent announcement by Francis C. Brown, president of the company.

The amounts given to independent research hospitals, institutions, and private research investigators more than double those granted during the first six months of 1957.

Principal expenditures went for additional research into projects on mental illness and arthritic and rheumatic disorders. Additional expenditures were made for new areas of research covering protein hormones, studies of blood chemistry, studies of the effects of various drugs on animals, and anti-shock drugs.

In addition to medical research, several grants in support of dental research on Schering's products have also been made.

Use of Ascorbic Acid in Meat Products.—The color in meat products is bound to the nitroso group, which is available as sodium nitrite or saltpeter. In manufacturing sausages, the addition of ascorbic acid reduces smoking time and the undesired multiplication of microorganisms which lower the keeping quality of the product. When properly used, ascorbic acid is not only harmless but reduces the amount of nitrite needed.—*Souci in Die Fleisch Wirtschaft (July, 1958): 458.*

Arsenic Poisoning in Man.– Arsenic poisoning generally escapes medical attention because the early signs and symptoms resemble those of infectious gastroenteritis. Diagnoses such as infectious diarrhea are found on the death certificates of patients with arsenic poisoning.

Arsenic is classified as a cumulative poison and is excreted in the urine and feces for some weeks after ingestion of a toxic dose. It appears in the urine within a few hours and rather simple sensitive toxicological tests can be readily performed in a laboratory to identify even minute amounts.—*J.Am.M.A. (July 26, 1958): 1688.*

for mastitis

. . . as an adjunct to proper herd management • Proven clinical effectiveness against the majority of causative organisms . . . staphylococci, streptococci, Escherichia and Pseudomonas . . .[1,2] *• Little or no development of bacterial resistance*[2]*— no antibiotic cross-resistance . . .*

FURACIN®

brand of nitrofurazone

Furacin has been shown to be strikingly effective in controlling mastitis under field conditions.[3,4] Of 7,123 lactating cows with acute mastitis, fair to excellent results were obtained in 5,597 (78%). Of 3,418 dry cows which had had mastitis during their previous lactating period, fair to excellent results were obtained in 3,104 (90%).[4] Furacin is highly effective also in combination with other antibacterials. In 7,104 quarters treated with Furacin-penicillin, ". . . marked improvement for the remainder of the lactation period, or complete clinical cures, occurred in 6,536 (92%)."[5] In another study with Furacin-penicillin, it is reported, "The total incidence of clinical mastitis in both herds has decreased to almost the vanishing point . . ."[1] These results have been confirmed,[6] and a subsequent increase in milk production and lack of any clinical signs of irritation have been noted.

FURACIN SOLUTION VETERINARY FURACIN 0.2% in aqueous solution—may also be used as a vehicle for certain antibiotics. Bottles of 500 cc. with rubber stopper, and 1 gal. **FURACIN-PENICILLIN GEL VETERINARY** FURACIN 2% and procaine penicillin (13,333 units per cc.) in peanut oil with aluminum stearate 3%. Rubber-capped vial of 100 cc.; applicator tube of 7.5 cc., box of 12.

Available through your professional veterinary distributor.

References: 1. Kakavas, J. C.; Roberts, H. D. B.; de Courcy, S. J. and Ewing, D. L.: J. Am. Vet. M. Ass. 119:203 (Sept.) 1951. 2. Kakavas, J. C.: Antibiotics Annual 1954-1955, New York, Medical Encyclopedia, Inc., 1955, p. 323. 3. Mires, M. H. and Chadwick, R. H.: Vet. News 10:3 (Jan.-Feb.) 1947. 4. Mires, M. H.: J. Am. Vet. M. Ass. 117:49 (July) 1950. 5. Mires, M. H.: Vet. News 14:9 (May-June) 1951. 6. Roberts, H. D. B.; Kakavas, J. C. and Biddle, E. S.: N. Amer. Vet. 34:247 (April) 1953.

NITROFURANS—a *new* class of antimicrobials—neither antibiotics nor sulfonamides

EATON LABORATORIES, NORWICH, NEW YORK

CLASSIFIED ADVERTISEMENTS

PERSONAL WANT ADS—$4.00 for the first 25 words and 10 cents for each additional word; 35 cents for use of box number.

TOTAL WORD COUNT must include complete box number address (8 words) or personal address line.

COMMERCIAL WANT ADS—$5.00 for the first 25 words, 25 cents for each additional word; $1.00 for use of box number. (See paragraph above for total word count.)

Remittance must accompany ad.

DEADLINES

1st of month issue — 8th of month preceding date of issue.

15th of month issue — 22nd of month preceding date of issue.

Names of classified advertisers using key letters can not be supplied. Address your reply to the box number, c/o JOURNAL of the AVMA, 600 S. Michigan Ave., Chicago 5, Ill., and it will be sent to the advertiser.

Wanted—Veterinarians

Veterinarian wanted—Practice 2/3 large animal; excellent facilities and equipment. Will consider 1959 graduates. Write A. W. Stults, 9 East Broad St., Hopewell, N.J.

Wanted—licensed veterinarian in eastern Iowa, large animal practice. Temporary salaried position. Address "Box T 2," c/o JOURNAL of the AVMA.

Veterinarian, Virginia licensed, to assist in mixed practice January through August. Opportunity for experience. Could lead to permanent arrangement. Address "Box T 3," c/o JOURNAL of the AVMA.

California licensed veterinarian for small animal hospital in Los Angeles area. Experience preferred. Dr. Mercer, 1120 W. Manchester, Inglewood, Calif., ORegon 8-8802.

VETERINARY NUTRITIONIST

Large pharmaceutical company near New York City offers a challenging opportunity to a person with commercial training and experience in the diseases and nutrition of large farm animals. He will be expected to assist in the survey, planning and coordination of new product possibilities as well as supervise research projects; also he will be expected to keep close check on nutritional and veterinary advances in the large animal field. D.V.M. degree plus training and experience in nutrition required.

Salary commensurate with qualifications. Excellent benefits program.

Please submit complete resume of training, experience and salary required. Address "Box T 11," c/o JOURNAL of the AVMA.

Assistant in small animal hospital, 20-year established practice. Volume assures experience for recent graduate. Must have Illinois license or be able to take examination in December, 1958. Suburb of Chicago. Address "Box T 14," c/o JOURNAL of the AVMA.

Veterinarian with Illinois or Missouri license wanted to assist in extensive small animal practice. Excellent facilities; living quarters if desired. Give full details. Address "Box T 10," c/o JOURNAL of the AVMA.

Wanted—Positions

Coming June graduate, University of Missouri, married, veteran, desires position leading to partnership with established general practitioner in Missouri or adjacent state. Address "Box T 1," c/o JOURNAL of the AVMA.

Relief veterinarian available for New York City and Long Island during last week of December and first week of January. Licensed and competent. Address "Box T 7," c/o JOURNAL of the AVMA.

Wanted—Practices

Desire to purchase established general or small animal practice in coastal California. Licensed, experienced. Address "Box S 23," c/o JOURNAL of the AVMA.

Wanted to purchase—established, lucrative, one- or two-man small animal practice. Desire information on practice in your original letter, gross, price. Address "Box T 5," c/o JOURNAL of the AVMA.

Experienced veterinarian desires purchase of small or mixed practice in Michigan or Wisconsin. Can pay $5,000 down. Address "Box T 8," c/o JOURNAL of the AVMA.

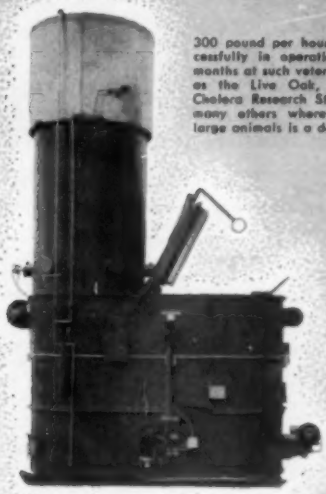
Licensed Florida practitioner, 35, married, wants lease with option to buy, rent, partnership, or percentage arrangement in small animal hospital. Address "Box T 12," c/o JOURNAL of the AVMA.

For Sale or Lease—Practices

For lease or for sale—new small animal hospital. Fast-growing section; excellent future; Arizona license required. Dr. S. Shapera, 4122 N. 36th St., Phoenix, Ariz.

ADDED!

TO SILENT GLOW'S COMPLETE ANIMAL CREMATORY LINE



300 pound per hour model, successfully in operation for many months at such veterinary stations as the Live Oak, Florida Hog Cholera Research Station and at many others where disposal of large animals is a daily problem.

the *NEW* 150 pound per hour model designed to solve your BIGGER and TOUGHER Disposal Problems

COMPLETE, SANITARY DESTRUCTION OF SWINE is one of the *toughest* animal crematory problems.

And this new, fully automatic incinerator solves this problem by destroying to a *white ash* swine and other large, disposal-difficult animals, contaminated laboratory waste and refuse . . . without smoke, odors, fly ash!

The 150 model and larger units in the line are so constructed that no leakage of liquids or fats can occur . . . *the animal fat actually is used as additional fuel*—just one of the several outstanding advantages offered by Silent Glow, leaders in the combustion field for thirty-five years.

MAIL COUPON TODAY!

Write for complete information on large and small equipment —a model for every requirement.

The **SILENT GLOW**
CORPORATION
868 WINDSOR STREET, HARTFORD 1, CONNECTICUT

☐ Send information on big equipment.
☐ Send information on smaller equipment.

NAME_____

ADDRESS_____

CITY_____ ZONE_____ STATE_____

For sale—Large animal practice in northern Illinois. Equipment and drugs. No real estate. Address "Box S 24," c/o JOURNAL of the AVMA.

Mixed practice with home and hospital for sale in Ohio to ambitious young veterinarian. Reason for selling: other interests. Address "Box T 4," c/o JOURNAL of the AVMA.

For lease—complete, new, small animal hospital with three-bedroom apartment attached. Located in large Middlewestern city. Address "Box T 6," c/o JOURNAL of the AVMA.

For sale—central Ohio mixed practice including hospital and home. Price of real estate; one-man practice. Address "Box T 9," c/o JOURNAL of the AVMA.

Established small animal hospital for sale in western New York. Low overhead; potential for large animal, state and federal work if desired. Modern, complete 40-cage hospital, colonial home. Price reduced for immediate sale. Address "Box T 13," c/o JOURNAL of the AVMA.

Virginia—established mixed practice in prosperous community. Reasonable price includes fully-equipped ranch styled small animal hospital and apartment, air-conditioning, two-way radios, etc. Address "Box T 16," c/o JOURNAL of the AVMA.

Too Late to Classify

Young, preferably experienced, veterinarian to cover small animal practice in Brooklyn, N.Y. Fully equipped, excellent opportunity. Salary, etc. on further request. Address "Box T 15," c/o JOURNAL of the AVMA.

Veterinarian wanted — Missouri-licensed for A.A.H.A. hospital. State all particulars in first letter. Address "Box T 17," c/o JOURNAL of the AVMA.

Index to Advertisers in This Issue

SAFETY FIRST...

with lasting protection

Swivax*

The superior hog-cholera vaccine for **SOLID** immunity...

MODIFIED LIVE VIRUS • RABBIT ORIGIN • VACUUM DRIED

*REGISTERED TRADE MARK

(Licensed under U.S. Patent 2518978)

PITMAN-MOORE COMPANY

division of
ALLIED LABORATORIES, INC.

all
my
nervous
troubles
are
well
in
hand

. . . and Jen-Sal® **DIQUEL** did it! The Diquel tranquilizing technic gives outstanding results that please doctors and clients alike. Expanded pharmacological activity offers these exclusive advantages: (1) **Tranquility of controlled duration** without side effects, (2) **Highly effective antihistaminic and anti-inflammatory properties** for better management of skin disorders, (3) **Greater antispasmodic activity** for control of nausea and vomiting. Order Diquel in two economical formulas — injectable for hospital use, tablets for dispensing.

Jen-Sal

Jensen-Salsbery Laboratories, Inc., Kansas City, Missouri